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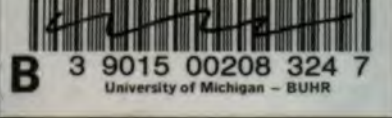
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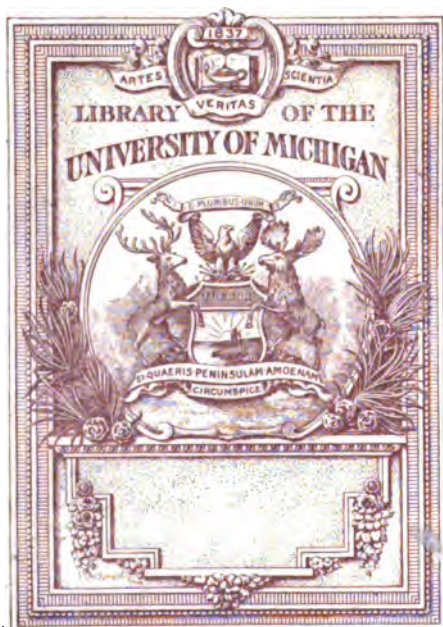
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Medicine.

THE MALADIES WITH WHICH TYPHOID FEVER MAY BE CONFOUNDED.

CLINICAL LECTURE DELIVERED AT THE CHARITÉ HOSPITAL, PARIS, FRANCE.

BY PROFESSOR POTAIN,

Professor in the Paris Faculty of Medicine; Member of the Academy of Medicine;
Physician to the Charité Hospital, etc.

GENTLEMEN,—The differential diagnosis in typhoid fever is important in Paris at the present time, where we are having this most unusual epidemic of the disease in the spring, which has already lasted two months,—February 15th to April 15th. There have been probably some three thousand cases so far, with a mortality of three hundred and thirty for Paris alone. We have nine cases in our wards to-day, and, though they are not of the very serious kind, they will do as a text to call attention to the different diseases with which they may be confounded.

In typical cases of typhoid fever you know that there is not much difficulty in making a diagnosis. The characteristic temperature variations, the patient with a whitish tongue and anorexia, his abdomen a little swollen, varying in sensitiveness in the right iliac region, with perhaps some rumbling sounds, while there is a little diarrhoea; added to all this you may have some sibilant râles in both sides of the chest, but no *souffle*. If these symptoms are present, there is hardly any room for doubt in the diagnosis. In the second week you may be more nearly certain, if you find some lenticular rose-colored spots on the abdomen or back, and you have, besides, prostration, even stupor, with a half-comatose state, insomnia, and a little delirium at night. It is then quite certain, and the third stage will give the fall by lysis, and the temperature will have the usual oscillations that are so well marked in typhoid fever.

But when these well-known and characteristic symptoms are not so well marked, it is quite possible that there may be errors, and many

good physicians will hesitate before pronouncing the dread name of typhoid. Let us see what the maladies are that may be mistaken for typhoid. One of the most common is ordinary gastric fever or bilious fever; indeed, in epidemics you will meet many cases that continue to have the regular symptoms of acute gastritis only. These fevers will have a regular rise in the temperature, rose-colored marks, and all the rest of it, and the differential diagnosis is not at all easy. "Grippe," or influenza, has also very similar symptoms to typhoid, for this infectious malady will have many varieties, according to the individual susceptibility or disposition. In "grippe" we often see the same depression and weakness leading to just as complete prostration as in typhoid, while the abdominal symptoms are quite similar, in some cases including swelling of the belly and noises in the right iliac fossa. There are some differences, but to catch them you must see the patient from the beginning of the attack and follow him up for days afterwards. At first the "grippe" will give intense headache, as a rule, and some shivering; this is in a person who has been almost well up to then. The commencement of typhoid is different, as here they will admit a loss of health for some time before they call you. The rise in the temperature will also be different; for instance, in "grippe" you may get 104° F. on the second or third day, while in typhoid you will not see this temperature until the fourth or fifth day, during which time the temperature will rise progressively.

In a certain number of patients we have seen the "grippe" and typhoid together, and again we have seen "grippe" provoke typhoid fever. In other cases "grippe" will commence the case, then give way to typhoid, and return during convalescence. It is then in its most serious form, for it will attack the nervous system and most likely carry off the patient. When the rose-colored marks are found it is certainly a typhoid, and as long as they are absent we are permitted to doubt its typhoid character.

There are two elements which I will insist upon that will allow you to always make the differential diagnosis. If the pulse is dicrotic, and this dicrotic element is accentuated, you may be sure it is typhoid (if the arterial pressure is low).

The tumefaction of the spleen is one of the more reliable signs in typhoid. It is true that it exists also in influenza in some cases, but it is much less marked. In typhoid it will be sixteen to seventeen centimetres, and in "grippe" it will not measure more than fourteen centimetres, and you know that the normal grand diameter is ten to twelve centimetres. One of these signs is not enough to make a sure diagnosis,

but when you get both of them you may be sure you are dealing with typhoid fever.

Another malady that sometimes takes on the same symptoms as typhoid is pneumonia. What is called pneumo-typhus comes mostly in those patients whose kidneys are insufficient; while they are capable of eliminating the excrementitious matters when in health, they cannot do so when overcome by an infectious disease that makes a superabundance of such material. This is why the pneumonias of old men are dangerous, because they have the typhoid form.

There are other causes for confusion and mistaken diagnosis here, for typhoid may commence with a pronounced predominance of pulmonary symptoms; chest dulness is present, and a considerable *souffle* is heard, and the mistake is easy to make. For that matter, pneumonia and typhoid can and do exist together, but this difficulty is not so great as it would seem to be. When the patient has passed fifty, for instance, it is pretty sure not to be typhoid, so that it is most likely a pneumonia that is giving the symptoms; besides, it is generally possible to find out if the patient has had a nephritis before, for you must not decide on the albuminuria present, as typhoid can give it. You will all observe albumin in typhoid, but it is a diffuse sort that does not precipitate; Professor Bouchard has shown that there is a renal form of typhoid that will give a retractile albumin that will precipitate with nitric acid. Finally, pneumonia does not often exist at the commencement of typhoid, so that it will not interfere with the differential diagnosis.

It is much more difficult to diagnose acute tuberculosis from typhoid; there are several varieties of acute tuberculosis, and the mistake is often made. In the first variety the thoracic symptoms may be very slight; there is a sort of typhoid state with prostration, vertigo, and noises in the ears, and here it is extremely difficult to make the differential diagnosis from typhoid. Just as in typhoid some subcrepitant and sibilant râles are heard, and as far as these signs are concerned the symptoms are identical. There are some little differences, however: the patient has a pulse in tuberculosis that is less dicrotic, while the arterial pressure is weaker and he has less anhelation or shortness of breath in typhoid. You must remember, though, that cases have been seen in the Necker Hospital that had no shortness of breath and presented all the symptoms of typhoid, and yet on autopsy there was a characteristic granular acute phthisis.

In these two maladies there is a prodromic period which has not the same form nor character. The prodroma of typhoid lasts for eight or ten days, and the patient has anorexia with considerable depression,

while his courage is gone ; but the prodroma in phthisis of the acute form is much longer : it will vary from three to six *weeks*, the patient will be weak and feeble, but continue to have great courage and hope. It is true that you may find some cases much quicker than this. I remember being called to see a girl who was still quite fat and looked almost brilliant, but had got thinner for some weeks back. The father was thin but in good health ; he stated, though, that he had had hæmoptysis as a young man. The girl's disease turned out to be acute tuberculosis.

Typhoid fever can also be confounded with another malady which has lately paid us a visit,—I mean exanthematic typhus fever,—but it shows a different evolution : its *début* is sudden and quick ; the typhoid symptoms are much the same,—weakness, stupor, and prostration ; though in the larger number of cases the abdominal symptoms are not so pronounced and the eruption differs markedly from the rose-marks of typhoid, so that slight attention to these points will prevent error.

There are still two maladies that certainly have not the reputation of resembling typhoid, but still they can assume its symptoms in the second period,—I mean syphilis and gonorrhœa. Just at the commencement of the second period of a syphilis there may light up a fever with the mucous plaques that will have all the appearance of typhoid ; but while there may be great prostration, and even stupor, there will not be any pain on palpation of the right iliac region nor any gurgling there. If there is a diarrhœa it will not be of the yellow ochre color of typhoid. Besides, you must look for the syphilitic eruptions, which, as you know, are sometimes very slight and not well known. It may even be that you will not find any skin manifestations, but you must hunt everywhere for the papules ; the anus, for instance, is much neglected, and yet it is here they will often be found ; a trace of the chancre may be found also, and the inguinal ganglions should be examined. This diagnosis is not difficult ; you must remember, however, that typhoid may exist with syphilis.

Gonorrhœa is still less difficult ; while there are cases that will have fever and much depression, even stupor, an exploration of the urethra will clear up the case. An acute nephritis might be mistaken for typhoid, but should not be confounded with it. A much rarer affection is pyæmia, but the repeated attacks of shivering that it gives rise to should make the diagnosis clear. Endocardiac infectious trouble resembles typhoid fever, but the cardiac signs, as well as the fact that the pulse is small and weak, while in typhoid it is full, will set the diagnosis right.

Other maladies are angiocholitis and cholecystitis ; the first is characterized by febrile movement at night and prostration, but the antecedent history will put you in possession of the case. As to the cholecystitis, it may be produced by calculi, and there may be a pericystic inflammation with peritonitis, which will pass on to the cæcum and produce a colitis accompanied by fever and gurgling in the right iliac region. The general symptoms may differ, but still they resemble each other enough to make mistake possible ; and, indeed, typhoid fever can cause cholecystitis. If the two diseases are present an incomplete diagnosis is quite possible ; still such troubles usually show themselves at a more advanced age than typhoid fever, but biliary troubles are seen now and then in young people.

Cerebro spinal meningitis is given as possible to mistake for typhoid, but I do not think it is possible to make the error. More often the diagnosis will be difficult with intermittent fever, but it does not often have the typhoid fever form here ; some of our patients have typhoid with an intermittence in the fever and a morning fall almost to the normal. The malarial element seems to impregnate some subjects and makes their typhoid disposed to febrile intermittences ; the appearance of the rose-colored marks will clear up this diagnosis. As to the continued fevers of hot countries of a malarial origin, we rarely see them in France.

ANÆMIA.

A CLINICAL LECTURE DELIVERED AT THE BUFFALO GENERAL HOSPITAL.

BY CHARLES G. STOCKTON, M.D.,

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CASE I.—This boy, aged eleven, had an attack of acute articular rheumatism two years ago, which lasted several weeks, and from which he has never fully recovered, having been confined to bed ever since. He entered the hospital a few days ago suffering from dyspnœa, occasionally amounting to orthopnœa, and it has been on account of this difficulty in breathing that he has been prevented from playing about as other children do. His bowels are regular, the urine is normal, he sleeps without anodynes, and has no pain when he keeps still in bed, but it hurts him to sit up. Previous to the attack of rheumatism he was well.

On inspection we notice that he is anæmic, that his respiration is rapid, and that the heart-beat can be readily seen. On palpation we find the apex-beat to be in the fourth intercostal space, but neither to the right nor to the left of its proper line. The impulse is accompanied by a very peculiar thrill. On auscultation I find a loud and hoarse systolic mitral murmur, which is transmitted to the axilla and to the back. The other sounds I cannot feel sure about. Those at the base are not heard clearly.

I have a theory by which to explain the phenomena of this case, as follows: The boy had rheumatism two years ago, and following this probably endocarditis, pericarditis, and pleuritis, which is not an uncommon combination of events. Since that time the anæmia produced by the rheumatism undoubtedly has continued, partly because of the rheumatic diathesis, partly because of his dyspnœa, poor oxygenation and nutrition, his close confinement and lack of activity. But how can we account for the peculiar behavior of the heart? It is found beating in the fourth intercostal space; it does not move from that level when the boy sits up; there is marked tympany immediately beneath, as if the

diaphragm with the stomach under it rose high. The respiratory murmur, while somewhat harsh, is clear enough to dissipate the idea of pulmonary disease. Now, what could make the heart stay in this false position and act in this excited way? The cardiac excitement might be accounted for by valvular lesions or by something wrong with the pneumogastric or the cardiac accelerator nerves. But these explanations would not account for the position, and we would expect the heart to be hypertrophied and dilated if the excitement were due to marked mitral regurgitation. The heart does not present downward and to the left as it would if there were hypertrophy or dilatation of the left ventricle. I believe that we have here an instance of what occasionally takes place, the adhesion of the heart to the pericardium and probably also the adhesion of the pericardium to the pleura, and, as a result of these adhesions, the heart has been held in its false position. Probably the adhesion began with the heart in its proper position, but as cicatricial contraction took place, the new-formed tissue shortened and drew the heart upward. This holding of the heart in a false position would irritate it continually, and give rise to tachycardia, to disturbed circulation, and, on exercise, to dyspnoea. I may change my opinion of the case on further observation. I am inclined to think I shall hold to my present view.

You will notice clubbed extremities in the boy's fingers, the somewhat pedunculated and pallid nails, the poor capillary circulation, all due, I believe, to the impaired general circulation and oxygenation.

Let me call your attention particularly to the matter of anæmia. It was probably in the beginning a toxic anæmia due to rheumatism, and it has been continued as a simple anæmia because the boy's respiratory power has signally failed, because his opportunities for exercise and active life have been removed. He should, however, be taken into the open air, have massage, inhalations of oxygen, and diet of the most nutritious character pushed to the limit of his digestion. With oxygen and food the child will have the blood that he needs. He requires exercise, but this he cannot take himself, and so it must be given him in the form of massage. I see no reason why the child should not become much better if the case is attacked vigorously.

CASE II.—This patient is a girl, twenty-three years old, a waitress, who has never been strong, but has been usually well until a year ago. The family history is not exactly satisfactory. The father died comparatively young, of a burn, and the mother died soon after the patient's birth. She has two sisters who are well and yet not strong. It is a rather doubtful family history, but it may possess certain pregnant facts

if we scrutinize it closely enough. The patient has suffered from dysmenorrhœa for several years, and has been decidedly constipated. Before coming here there was marked menorrhagia for several periods. She has been treated for this since coming into the hospital, and is now much better, but still suffers from the anæmia, as is very evident. She complains of palpitation of the heart and insomnia. She feels sleepy during the day when she is sitting up, but when she lies down she cannot go to sleep.

I find no evidence of cardiac disease. The heart is in its proper position, of proper size; the sounds are normal, although there is the suspicion of a bruit, which I should probably find distinct if she were lying down, and which is undoubtedly an anæmic bruit. Originally the case was probably one of chlorosis,—that is, an anæmia characterized more by the loss of hæmoglobin than by the diminution in number of red blood-corpuscles. The yellowish-green appearance of the skin, which is characteristic of chlorosis, is not marked at present, doubtless because she has been under treatment for some time. The continuance of her menorrhagia leads me to think that there has been some connection between her menstrual difficulty and the anæmia, although in chlorosis there is usually amenorrhœa.

To estimate the quantity of hæmoglobin, I use the hæmatospectroscope of Hénoc. This instrument is based upon the fact that of normal blood a definite thickness obscures the light, and that, as the proportion of hæmoglobin decreases the thickness of blood necessary to produce the opacity becomes greater. Between two slips of glass which are in contact at one end, but which diverge towards the other end, I let the blood flow from a needle wound in the girl's finger. Along the larger of the slips of glass is an index marking the point at which the light is obscured by normal blood, and with a numbered scale from which, by reference to the accompanying table, the proportion of hæmoglobin can easily be ascertained. With the instrument there is also a spectroscope for noting the bands of the spectrum of oxy-hæmoglobin. In normal blood I could see the bands most plainly at the point marked 20 on the scale. I am in this case able to see the bands at 20, 30, 40, and even at 50, but they are seen most plainly at 44. By reference to the table I find that this indicates the presence of only forty-five per cent. of the normal amount of hæmoglobin. Although you see considerable color in the lips and a pinkness of the ears, yet these must not deceive you in regard to the presence of anæmia. Although the lips are red, inspection of their inner surface and of the gums reveals pallor.

I have constantly impressed upon me the fact that there is a relation between indigestion and anæmia, and that persons suffer from indigestion not only on account of taking improper amounts and kinds of foods, but because the food is improperly treated in the stomach. Although there may be sufficient nutriment taken and no complaint referred to the stomach, there may be present such a loss of function on the part of the stomach that the decreased absorption of nutriment cannot be compensated for farther down in the alimentary canal, and anæmia succeeds. The insufficient absorption of nutriment is not the only factor in the relation of indigestion to anæmia. Nature provides in the alimentary canal two great means of producing an aseptic condition of the contents of the digestive tubes. The stomach is likely to be the seat of putrid fermentation unless kept aseptic by some substance, and this is supplied normally by the secretion of hydrochloric acid; but if there is a lack of hydrochloric acid, putrefaction is apt to occur.

Even if there are no evidences of dyspepsia, a lack of hydrochloric acid implies some degree of indigestion, and the food goes down into the intestinal canal so imperfectly changed that the second great means naturally supplied to keep the contents of the alimentary tube aseptic is insufficient. I refer to the bile. Its secretion is stimulated in large part by the presence of hydrochloric acid. When, therefore, there is not a proper secretion of hydrochloric acid, the bile does not appear in sufficient quantity to keep the intestinal contents in a fairly aseptic condition. Thus from faulty gastric digestion, and particularly from insufficient secretion of hydrochloric acid, there results a more or less septic state of the whole alimentary canal, and from this there very easily follows a toxæmia. Toxæmia I believe to be a very important factor in the production of anæmia, and this accounts for the fact that some persons who eat largely still suffer from anæmia, because the food, although taken in sufficient quantity and of proper quality, cannot be absorbed as nutriment; or, if absorbed, in the presence of toxæmia, assimilation fails. In the present case we have a faulty appetite to deal with, an appetite for bread but not for meats. I should naturally think that this girl was lacking in the hydrochloric acid secretion, and I believe that the anæmia and her subsequent ailments have arisen from imperfect digestion, which is at the bottom of more cases of anæmia than any other factor.

CASE III.—This girl has been in the clinic before on account of rheumatism and cardiac disease. This morning she is brought before you as another lesson in anæmia and the relation which rheumatism

bears to that affection. She was quite well before her rheumatism, but since then she has been ill on two accounts: first, because she has not had enough blood; and, second, because she has not been able to utilize the blood which she has.

A person with anæmia, but with sound organs, will shortly be able to overcome the condition, for the blood-making power under such conditions is prodigious; but when there is (1) a faulty digestive tract, (2) a faulty respiratory apparatus, (3) and a faulty circulatory apparatus, the anæmia is very apt to be obstinate. This patient has a failure of digestive, of respiratory, of circulatory power; her nervous system is depressed, and, in fact, her functions have all been impaired. Her heart has rendered it impossible for her to use to advantage the blood that she has. The respiratory power in this case is greatly diminished by mechanical means due to the failure of the circulation. With her poor circulation she cannot have a proper digestion. As an illustration of the condition of the circulation, look at her hand, mottled blue and white, without a trace of natural color. When you last saw her it was necessary to bring her into the room on a stretcher, but she is now much better, and the reason for her improvement is, I think, a compensatory hypertrophy of her left ventricle. Her heart, although still weak, is stronger, her circulation is becoming better, and the girl is getting into a condition which will enable her to make blood. She ought to be liberally fed; she should have massage; she should be removed from the hospital and its atmosphere of sickness if she had a good place to which she could go.

In addition, great attention must be paid to the condition of the blood. In intense anæmia there is usually a lowered alkalinity of the blood; there is an excessive amount of uric acid in the urine which may be taken as an index of the increased amount of uric acid in the blood. This state of the blood has been called by Von Jaksch, uric-acidæmia, and is a condition very common indeed in anæmia. Practically you will be unable to derive full benefit from the administration of iron or other agents that improve hæmatisis, unless you give alkalis sufficient to bring the alkalinity of the blood up to the standard. In this girl's case the alkalis may seem of special use because of her rheumatic diathesis, but they are of even greater value because of the importance of raising the alkalinity of the blood. In all cases of anæmia, therefore, I advise you to examine repeatedly the urine as to its reaction, not weekly nor daily, but several times in the course of the day, and administer an alkali, not in doses large enough to keep the urine alkaline, but so as to prevent hyperacidity. I have seen a

good many cases of anæmia improve upon alkalies alone, without any iron, arsenic, cod-liver oil, and without particular attention to the diet. The alkalinity of the blood is a very important matter and one that receives less attention than it deserves. The administration of iron is simply the A, B, C of the treatment of anæmia, and is familiar to every one, even including the laity. You must not alone understand the alphabet of this treatment, but something higher, and must learn to manage the economy so that the iron will accomplish your object; and if the iron seems to harm the patient, as occasionally happens, you must be able to treat the anæmia without the use of that agent.

CASE IV.—Here is another case of anæmia which you have seen before on account of another trouble. The patient is a woman of sixty-six. She has a broad, pale, very smooth tongue, robbed of its epithelium, indented by the teeth, slightly fissured, tremulous. It is the index of cachexia and lowered vitality. You will notice the extreme pallor of the face and its somewhat icteric color. The skin has also rather a waxy appearance. There is emaciation and evidence of a trouble which I have described to you before. Among other things, you will see that the patient is extremely anæmic. I will not subject her to the annoyance of drawing a drop of blood and examining it in your presence, but I will assure you that her blood contains less than fifty per cent., and, probably, not more than forty per cent. of the normal amount of hæmoglobin. The patient says that she feels better than she has felt before in a year. [Patient removed.] She is one of the kindest and gentlest patients we have had in the hospital this winter,—always getting better, always making the best of everything. When we consider that she is an old woman suffering from a carcinoma of the omentum, and probably, also, of the cardia, with profound anæmia and insomnia, and that, in spite of all her troubles, she is kind and gentle, it ought to make the majority of us ashamed of ourselves. I believe that her happy disposition is one reason why she feels as well as she does. If she were one of those patients who struggle against all the hardships that come along in such an illness, she would have been dead long ago. Many comparatively healthy people virtually take their lives struggling against what they cannot overcome. This patient is really not very much weaker than when I first presented her before you. She is surely and steadily growing worse, though not very rapidly.

The interesting point about the case this morning is its relation to the general subject of anæmia. Here, however, you see the anæmia of chronic and malignant disease. Nothing can overcome it. The

administration of iron is useless to such a patient when the poison of the disease is constantly defeating your efforts. All you can do is to give foods to nourish the patient as much as possible; to provide an abundance of oxygen, pure air, and await the coming of the event, which will not be slow.

This morning we have studied a variety of causes of anæmia, dwelling more at length upon the anæmia due to rheumatism, and considering also the anæmia from indigestion, intestinal toxæmia, and, last, the anæmia from chronic malignant disease. In all of these cases it is important to treat the prominent symptoms of anæmia, and yet, to overcome the anæmia, you must overcome the disease to which it is due. How are you to overcome the anæmia of the chlorotic girl if the essential cause of the chlorosis remains? You must keep her bowels open, for it is useless to give iron to a chlorotic girl if the absorption of the toxic principles from the intestines continues. Again, when the anæmia is due to faulty digestion and imperfect assimilation of nutriment, it is of little use to give iron until the digestion is improved. In the case of rheumatism, it is of no use to give iron unless the rheumatism and the excess of uric acid are overcome. So, too, in this last case, it is useless to attempt to overcome the anæmia, for we cannot remove the cause; we can only relieve some of the more distressing symptoms of malignant disease by giving anodynes.

MITRAL STENOSIS: ITS DIAGNOSIS, RESULTS, AND TREATMENT.

CLINICAL LECTURE DELIVERED AT GUY'S HOSPITAL.

BY FREDERICK TAYLOR, M.D., F.R.C.P.,

Physician to, and Lecturer on Medicine at, Guy's Hospital.

GENTLEMEN,—I wish to-day to draw your attention to a case of mitral stenosis which has recently been under my care.

H. W., aged twenty-two, was admitted on January 19, 1894, for palpitation of the heart and pains in the chest. At eight years of age he was under treatment for spinal curvature and cervical abscess, and then caught scarlatina. He has never had rheumatism. He has been in the hospital three times previously. In January, 1891, he came in with dyspnœa and cough, diffused cardiac impulse, an apical systolic and a presystolic murmur. He was relieved in a fortnight; three weeks later he was admitted and stayed two months. In March, 1892, he was in again with præcordial pain, dyspnœa, and palpitation; the murmurs were a systolic apical murmur and a presystolic murmur alternating with a mid-diastolic.

Since his discharge he has done very little work, and in the summer of 1892 he began to have, on exertion, attacks or "fits," in which he had throbbing pain in the epigastrium, and then became unconscious for a few seconds. These attacks have continued ever since. For the last six months he has had œdema of the legs.

On admission he has a moderate degree of dyspnœa and his face is somewhat dusky and congested. The cardiac impulse is somewhat diffused; it can be felt best in the sixth left interspace in the nipple-line, and there is an obscure thrill felt over it. The præcordial dullness is somewhat extended to the left. A harsh blowing murmur replaces the first sound, and is traceable into the axilla in one direction and as far as the right nipple in the other. It is only faintly heard behind. A presystolic murmur can be heard in the axilla two inches outside the nipple. The second sound at the base is accentuated. The pulse is

ninety-two, full, and regular. There are a few râles in the chest. The liver is enlarged, reaching two inches below the costal margin; the spleen is not enlarged. The urine is of specific gravity 1024, and contains no albumen. He was kept in bed, and was ordered a diuretic mixture containing potassium acetate, squills, broom, and spirits of nitrous ether. As the pulse was regular and not very rapid, no digitalis was given. For a time he improved somewhat, but he had several attacks of præcordial pain and palpitation, in some of which he became unconscious for a time. Early in February he had tonsillitis with some pyrexia, but on the 12th he was well enough to be allowed up for an hour or two. A week later he had pains in many joints, frequent attacks of cardiac pain and palpitation, thoracic pain resembling that of pleurisy, occasional vomiting and increasing prostration, with rapid breathing and quick, feeble pulse; but the latter was always regular. The systolic murmur persisted throughout, audible right across the base of the chest in front from the right nipple to the axilla, but never loudly, and sometimes it was not heard at all behind. The murmur of diastole varied a good deal; sometimes it was described as presystolic, at others as mid-diastolic; at others, again, it was a continuous booming sound which appeared even to occupy more than the diastole, and extend over the whole period of revolution of the heart. It was always heard best at the front border of the axilla, one and a half to two inches external to both the nipple-line and the impulse. Towards the end a definite systolic thrill was felt between the nipple and the sternum and the liver pulsated very distinctly. The urine continued to be high-colored and deposited urates, but it never contained any albumen. He died on February 20. At the post-mortem examination the heart was found to weigh eighteen ounces; the right ventricle occupied nearly the whole front of the heart and was greatly hypertrophied. Its wall measured seven-eighths of an inch in thickness. The right auricle was also hypertrophied and dilated. The tricuspid orifice measured five and a half inches. The left ventricle was very small, and its wall measured less than one-half of an inch in thickness. The orifice of the mitral valve would only admit the tip of the little finger, and measured one and a half inches in circumference; the curtains were much thickened and extremely hard; the chordæ tendinæ were short and very thick. The aortic valves were healthy.

The lungs were in a condition of red induration, and there were old filamentous pleuritic adhesions on the posterior surfaces, but no recent pleurisy. The pulmonary arteries were thickened and rigid. The liver was congested,—“nutmeg”; the kidneys were not examined. The

condition was, therefore, one of mitral stenosis, with tricuspid regurgitation, congestion of the lungs, liver, and probably of the kidneys.

Now, gentlemen, to go fully and minutely into the whole subject of mitral stenosis, with illustrations from clinical cases, would, I fear, take far more than the time allotted for one of these discourses. I shall, therefore, confine myself to a small number of points which this case specially illustrates, or in which this case differs from the usual rule.

The Murmurs of Mitral Stenosis.—I do not say the *murmur* of mitral stenosis, but the *murmurs*, because it is possible to hear more than one kind of noise as the result of this lesion. In the case of H. W., two murmurs were audible at the apex: one was a systolic murmur which, with certain reservations, I may say no one would say was due to the stenosis; the other was a loud rumbling sound heard during diastole, and, as a rule, filling up the whole of the interval between the second sound and the succeeding first sound. In the proper sense of the word, as it seems to me, it was a diastolic murmur,—that is, it was a sound occupying the period of dilatation or diastole of the ventricles. If I were to ask a number of you what is the murmur of mitral stenosis, I believe I should get from the greater number the answer,—a presystolic murmur. To my mind that is an inadequate answer. What is a presystolic murmur? It is a murmur occupying the diastolic period between the second sound and the succeeding first sound, and, therefore in that sense also a diastolic murmur, but distinguished from other diastolic murmurs by often commencing at a short interval after the second sound, and by running up to—that is, getting louder as it approaches—and finishing off with the succeeding first sound. You must understand that the terms systole and diastole in relation to murmurs refer to the ventricles; that the period of the heart's revolution, as it is called, is equally divided between the ventricular systole and ventricular diastole, and that, therefore, any murmur which is not systolic must be diastolic, and any which is not diastolic must be systolic. There is no room for a presystolic murmur, except as a special form of a diastolic murmur. Now, as a fact, the ear can recognize in different cases three forms of diastolic murmur: one which begins with the beginning of diastole—that is, with the second sound—and lasts a variable time, either fading away in the middle of the period or running right through, but never getting louder as it approaches the next first sound; another which begins distinctly after the second sound and similarly fades before the succeeding first sound; and a third which, beginning after the second sound, runs up to and intensifies with the occurrence of the first sound. I think the best nomenclature for these is that pro-

posed, I believe, by Dr. Bristowe,—namely, to call them respectively (a) early, (b) mid-, and (c) late diastolic. The other possible names are, respectively, (a) diastolic or pure diastolic, meaning that it commences accurately with the second sound, (b) post-diastolic, and (c) presystolic. To presystolic there is no objection, but the other terms are only justifiable on the assumption that the term diastolic in this case refers to the diastolic or second sound, and not to the diastole or state, and, therefore, period of dilatation of the ventricle.

I have said I thought "presystolic" was an inadequate answer to the question proposed. It is for this reason that under different circumstances—that is, in different stages—you may get as a result of mitral stenosis any one of three murmurs above mentioned. Why is this? Well, you must bear in mind that the mere presence of a narrowing in a given channel does not bring about sound: there must be some fluid passing through it with sufficient velocity to make audible vibrations either in the edges of the orifice or in the fluid on the far side of the orifice. The forces driving the blood through a stenosed mitral orifice are the passive flow of blood from the pulmonary veins through the auricle and the contraction of the auricular wall itself. Both of these forces may vary very much: the presence or absence of regurgitation will alter the force of the direct blood-flow; the amount of dilatation or hypertrophy of the auricular wall will give different results in the sound which its contraction produces by driving blood through the narrow orifice. Now, this last factor especially is likely to be affected by the stages of the disease. In the first stage the auricle is likely to be still powerful, either with its natural powers of contraction or with a force increased by hypertrophy; in the later stage dilatation will have proceeded to an extreme, and the auricular force of contraction will be correspondingly diminished. The presystolic murmur has long been, and I believe justly, attributed to that natural contraction of the auricle which takes place during the latter portion of diastole and immediately precedes the ventricular contraction. You would, therefore, not be surprised to hear a well-marked presystolic murmur in the early days of mitral constriction; on the other hand, in the later stages, when the auricular force declines, the presystolic murmur might be expected to lose its importance.

Now, as a fact, this is what to a great extent does occur. One can, I think, recognize three stages in the course of a case of mitral stenosis: a first stage when there is a typical rumbling presystolic murmur, when the heart is acting well, fully compensated or nearly fully compensated, the patient having a quiet, steady pulse and suffering few

symptoms, perhaps only a little shortness of breath on exertion. These are cases of which you will see more instances among the out-patients than in the wards. They have mitral stenosis, but they are yet in an early stage and not bad enough to want to come in.

In a later stage the presystolic murmur in its typical form is no longer heard, or heard only from time to time, and in its place is audible that murmur which begins after the second sound, but passes away, often leaving a long pause before the next first sound. It is the mid-diastolic murmur. Now, there are three features about this that are of interest. The pause I have just mentioned is liable to be obliterated if the heart beats very quickly; often it is irregular; three, four, or five beats follow quickly one upon the other, then comes a beat or two beats more slowly. In the slow beats there is a pause, and the murmur does not touch the succeeding first sound; in the quick beats, though the murmur reaches the first sound, there is no intensification or greater loudness, and the resemblance to a presystolic murmur is only partial. Secondly, the murmur commences in what appears to be the second half of a reduplicated second sound. Thirdly, there is practically, I think always, a systolic murmur as well.

At this stage the heart-sounds are liable to vary; under certain circumstances, often such as are equivalent to increased vigor of the action of the heart, the presystolic murmur of typical character will return and replace the mid-diastolic, and again it will disappear and the mid-diastolic assert itself. These changes may be very rapid; one of the murmurs may persist only for a few minutes, a few hours, or for some days. This is not unintelligible on the assumption that the driving force is responsible, seeing that even in health there are great variations of the actual force of contraction of the heart. Patients in this stage form a large proportion of the cases of mitral stenosis we see in the wards.

A third and generally very late stage, though one from which temporary recovery is possible, with the result that the patient goes through it all again on a subsequent occasion, is one in which the heart is beating very quickly, very irregularly, tumultuously, and giving the impression that it is tumbling about almost anyhow. A somewhat humming or half-musical rumbling seems to occupy the whole period of revolution, but at intervals—that is, with each beat of the heart—a high-pitched systolic murmur is heard. Now, I suppose there can be no doubt that the rumbling noise is a prolonged diastolic murmur, but it is extremely difficult to discriminate its exact relation to the impulse or to the sounds, and the systolic murmur, which can be identified as such, *seems* to overlap it and not to be distinctly separated from it. At this stage the

patient is generally very bad, with dropsy, dyspnoea, œdema of the lungs, albuminuria, and all the advanced results of stenosis of the mitral orifice and its complication, tricuspid regurgitation. If improvement takes place the heart becomes slower, and often the murmur then assumes the character of the mid-diastolic, and less often of a typical presystolic murmur. In the case that I have taken for my text to-day there was an approach to this kind of murmur; it may be described as something intermediate between a presystolic and the extensive rumbling, wholly diastolic, I have just spoken of.

These, however, do not exhaust the possibilities of the behavior of the heart with regard to murmurs in mitral stenosis. In a few cases there is an early diastolic,—that is, a murmur beginning with the second sound and ending in the middle or nearer the end of the diastolic period. Such a murmur is, I think, always preceded or accompanied by a systolic murmur, and is usually of shorter duration than this systolic murmur. The two together closely resemble the double murmur so commonly heard at the base of the heart over the aorta, and constitute, indeed, a real *to-and-fro* combination, which is the case with none of the sounds that I have so far spoken of. It has been suggested that the mechanism is as follows: blood is regurgitated through a stenosed orifice into the left auricle, which becomes so distended that on ventricular relaxation the excess under high tension is returned with sufficient vigor in the first part of the diastole to produce a murmur. In the latter part of the diastole we must suppose that neither the passive flow of blood from the lungs nor even the contraction of the weakened auricle is adequate to produce audible vibrations.

Then, lastly, there may be no murmur at all, either diastolic or presystolic. This occurrence is distressingly frequent. What is the explanation of this? I think I have already suggested what it may be: a sound is not due to the instrument alone; the instrument must be played upon. There must be the pipe, or the strings, or the membrane on the one hand, and there must be the wind, or the violin-bow, or the drum-sticks on the other, and these latter must be driven or manipulated with the force proportionate to the construction of the former. In a certain number of cases of mitral stenosis in the very last stages, when, for instance, patients are brought moribund into the hospital, the diastolic murmur may be absent; it is probably the result of the absence of force either in the passive blood-flow or in the auricular contraction to produce the necessary vibrations.

Only a short time ago a patient came in who proved to have mitral contraction. She was under our observation only one month before

she died. No murmur of mitral stenosis was audible ; but what murmurs she may have had in past times I cannot, of course, say. I remember a patient long ago, who came in moribund from cardiac failure, and without any characteristic murmur ; after death the left auricle was found full of ante-mortem clot, leaving only a narrow canal through which the pulmonary blood found its way into the left ventricle. But there are other cases in which, for periods of months, in varying times of great difficulty and comparative health, no diastolic or presystolic murmur is ever heard. F. H., another sufferer from mitral disease, first came under our observation on August 10, 1892, and died on February 20, 1894, a period of eighteen months and ten days, or say seventy-eight and a half weeks. Out of that period he was in the hospital fifty-nine weeks ; he had throughout a systolic apex murmur ; but though a mid-diastolic murmur was occasionally heard in the first few months of our observation, no murmur of the kind was ever heard in the last fourteen months of his life. Nevertheless, after death there was found a button-hole mitral, of which the orifice would scarcely admit the tip of the little finger, and measured less than one and a half inches in circumference,—that is, much less than half an inch in diameter, supposing it had had a circular shape. As it was not circular but elongated, the area for the passage of blood was still less than the diameter of a half-inch corresponds to. I know the question that all this leads up to. It is, How is a diagnosis to be made? How is mitral stenosis to be recognized? Well, first, you must recognize that mitral stenosis is extremely frequent ; that the majority of cases that die of mitral disease have mitral stenosis by the time they do die, whether typical murmurs have been heard or not. Secondly, if you can exclude aortic regurgitation from your case, any murmur occupying any portion of the diastolic period may be taken to result from mitral stenosis. Time does not allow me to go into the special reasons for making this reservation. Thirdly, you must always be suspicious of mitral stenosis in cases where there is a systolic murmur inaudible behind, where there is in association with it a reduplicated second sound, and where there is evidence of tricuspid regurgitation.

Position of the Murmur.—It is a point of some little importance to remember that a presystolic murmur is nearly always best heard, and it may be alone heard, at the very point on the surface of the chest at which the impulse takes place. If you wander half an inch from that point you may fail to hear a presystolic murmur. In the present case the murmur of mitral constriction was heard nearly two inches to the left of the nipple. I think this is probably explained by the high degree

of hypertrophy of the right ventricle, which compelled the left ventricle to keep quite in the background, and hence the vibrations produced in its cavity could only reach the surface farther back than usual.

Thrill.—Some abnormal sensation was felt over the præcordial area shortly after admission, but it was only a few days before death that a definite thrill was felt, and this thrill was systolic in time (*frémissement cataire*) and occupied a position midway between the sternum and the nipple. Now, has a thrill any significance? Is it of any value in diagnosis? A common answer to this question is, that "a thrill means mitral stenosis." This is a very unsatisfactory answer. It is correct only up to this point, that in the majority of cases where a thrill is felt there undoubtedly is mitral stenosis; but even when it is present under these circumstances, it is often not the direct but only the indirect result of the mitral disease. You must remember that a thrill is caused by certain vibrations being palpable as well as audible. The majority of cardiac murmurs that we hear are audible only, and their vibrations are of such a kind—that is, of such high pitch or of such little amplitude—that they are not appreciable by the coarser nerves of touch, but only by the more highly specialized nerves of hearing. But, nevertheless, it is conceivable that at every one of the cardiac orifices, both with systole and diastole, there should sometimes occur vibrations which are palpable and productive of thrill, as well as audible and productive of murmur. I have felt thrills in connection with the following murmurs: systolic mitral, diastolic and presystolic mitral, systolic aortic, diastolic aortic, systolic tricuspid, and systolic pulmonary; and I believe I have felt a tricuspid presystolic thrill. That makes seven out of the eight cardiac murmurs; and the eighth, a pulmonary diastolic murmur, is so rare that it is excusable if one has not met with a case of any kind, much more a case in which a thrill was present.

There are, moreover, three other conditions which lead to palpable vibrations or thrills either over or near to the præcordial area. One is an aneurism at the root of the aorta; another is a communication between the aorta and the pulmonary artery, generally, however, the result of an aortic aneurism; the third is a communication between the two ventricles by a perforation through the septum ventriculorum. So that you see the bare statement that a thrill exists tells you no more really than the mere announcement that there is a murmur. If you are told of a thrill in the same way as if you are told of a murmur, you must know in what part of the præcordial area it is found, and by careful observation it can be localized just as accurately as can a murmur; and you must know what part of the heart's revolution, systolic

or diastolic, it occupies. As a fact, the most common thrill is undoubtedly a presystolic mitral; a very large proportion of presystolic typical murmurs are accompanied by thrill; mitral regurgitation is rarely so accompanied. I know that regurgitation through a tightly-stenosed orifice may produce a systolic thrill; and if this is frequent, it is a case in which thrill is *indirectly* due to stenosis. Thirdly, tricuspid murmur is rather frequently accompanied by thrill, and tricuspid dilatation or regurgitation are in a very large number of cases the result of mitral stenosis. This last is the case we have before us: a patient suffering from considerable mitral stenosis; tricuspid regurgitation shown by the systolic murmur audible up to the sternum and by pulsation of the liver, and a well-marked systolic thrill coming out over this area between the impulse and the sternum during the last few days of life.

I leave open the question, What are the exact physical conditions of the orifice or of the driving force which cause the vibrations to be palpable as well as audible? They have to be determined before one can speak with confidence of the significance of thrills, as different from murmurs.

Pulsation of the liver is the next feature in this case that I wish to draw attention to. Physiologically and clinically this is a very interesting phenomenon, and one that is a great deal more common than has sometimes been supposed. I remember that a few years ago the question of its rarity was raised, and I was able at that time to point to five cases which I had had under my care within six months in this hospital; and now within ten days there have died in my wards three cases of heart-disease with pulsating liver; two of these were cases of mitral stenosis, the third was one of aortic disease with mitral regurgitation.

I think it only wants to be looked for, or rather felt for, to be found; not by any means always, but in a large proportion of cases of mitral stenosis in the later and latest stages. If the liver is enlarged, as it so often is, to a great size, and if there already exists a tricuspid murmur, place the hand firmly on the organ and ask the patient to hold his breath for a short time. You will then, in these cases, feel a pulsation often irregular in force and time, as that of the heart is, but often much more extensive in its excursions than the impulse of the heart on the side of the chest. In the most pronounced cases, by placing one hand in front and the other behind under the right twelfth rib, not only can pulsation be felt by both, but a pulsatile expansion of the organ can be felt, showing that it is really due to a force arising from within it, and is not a mere shock or elevation resulting either from the heart, the aorta, or the vena cava. The mechanism is appar-

ently simple: in consequence of the yielding of the tricuspid valve, blood is driven during ventricular contraction from the right ventricle into the right auricle; the large opening of the inferior vena cava into the right auricle is close to the tricuspid orifice; the opening of the hepatic vein trunk into the inferior vena cava is close to the opening of this vessel into the right auricle. The flow of blood into the liver by this means is also aided by gravity. That the force of the right ventricular contraction should be felt in the hepatic vein and its branches under these circumstances is, therefore, not surprising. With a little trouble a tracing can be taken from the surface of the abdomen over the liver either with a cardiograph or even an ordinary sphygmograph, and such a tracing will show a close resemblance to the tracings taken from the pulsating jugular vein under similar circumstances.

Albuminuria.—It was a somewhat unusual feature in this case that in the five weeks which preceded his death, and during which he was under our notice, no albumen was found in his urine. We know that the common result of the stagnation in the venous system that follows mitral stenosis is considerable congestion of the kidneys and passage of albumen into the urine. The post-mortem examination was only partial and the kidneys were not examined; but the obviously slight implication of these organs in this case is probably to be explained by the fact, which we are often compelled to recognize, that in different cases of heart-disease the organs or systems liable to suffer from the general venous stagnation may be very differently affected. Thus, in one case there may be much general dropsy; in another much œdema of the lungs without œdema of the legs; in another much enlargement and pulsation of the liver with little or no anasarca; in another, finally, every organ, as well as the cutaneous system and peritoneum, may show the extreme effects of hinderance to the venous circulation.

Treatment.—Time does not allow me to open the question of treatment, and I will only here remind you of the general principles which you should bear in mind when you have to deal with a case of mitral valvular disease with failure of compensation. First, you must insist on absolute rest; secondly, you must prevent any overloading of the vascular system, both by moderation in diet and by the use of laxatives, diuretics, and such drugs as will keep the secretions active,—in certain cases even venesection may be desirable; thirdly, you will in suitable cases use such drugs as digitalis, strophanthus, and others which have the effect of strengthening and steadying the action of the heart; and, fourthly, you may have to deal with special symptoms, such as pain, cough, sickness, sleeplessness, which may arise in the course of the illness.



ADDISON'S DISEASE. DR OLIVER'S CASE Newcastle-upon-Tyne

ADDISON'S DISEASE AND ITS TREATMENT BY SUPRA-RENAL JUICE.

CLINICAL LECTURE DELIVERED IN THE ROYAL INFIRMARY, NEWCASTLE-ON-TYNE.

BY THOMAS OLIVER, M.A., M.D., F.R.C.P.,

Physician to the Royal Infirmary, Newcastle-on-Tyne, and Professor of Physiology
in the University of Durham, England.

GENTLEMEN,—I bring before you a patient who is the subject of rather an interesting malady,—viz., Addison's disease. Apart from the interest attaching to the disease itself, it has for us who are living in the North of England a special attraction. Many of you may not be aware that it was in this locality the physician was born who subsequently lent his name to this morbid condition. Dr. Thomas Addison was born in Long Benton village, three miles from Newcastle. He was the son of a small grocer. Though born in rather humble circumstances, his father sought for his son the best instruction he could secure. Young Addison had for his first teacher John Rutter, the parish clerk at Benton, by whom, too, Robert Stephenson was taught, the son of George, the inventor of the locomotive. Later on he was transferred to a school at the Three-Mile Bridge, then to Newcastle, and subsequently, as his father's means were increasing, Benton having become a prosperous village through the development of the collieries in the neighborhood, young Addison was sent to Edinburgh, where, having completed his primary education, he enrolled himself as a medical student of the University, ultimately receiving in due course his doctorate of medicine. Thus qualified he made his way to London, became a pupil at Guy's Hospital, and afterwards one of its physicians. It was at Guy's, after being thirty years on the staff, that he discovered the malady to which Trousseau, when subsequently describing it, applied the term Addison's disease.

Addison had been struck by the existence of a peculiar form of anæmia without discoverable cause. He had noticed that it pursued in every instance a similar course, that it extended over a lengthened

period, and ultimately terminated fatally. As the accompaniments of this general anæmia, he observed an increasing languor and debility, extreme feebleness of the heart's action, irritability of the stomach, bronzing of the skin,—more marked in certain places than others,—symptoms with difficulty explained, but found almost invariably associated with a diseased condition of the supra-renal capsules. I have said that practically Addison was a Newcastle man. It is a happy coincidence that one of the best monographs on Addison's disease is the published Croonian Lecture by the late Dr. Greenhow, of the Middlesex Hospital—a nephew of Mr. Greenhow, formerly surgeon to this Infirmary.

As our patient presents in a striking manner all the physical signs and symptoms of Addison's disease, I cannot do better than give you a few notes of her case.

Elizabeth D., aged twenty years, duster in a pottery, was admitted complaining of increasing weakness of six months' duration, of discoloration of the skin, of vomiting, and of having suffered from pain in the epigastrium. Her father and mother are living and are healthy. She is one of a family of thirteen, of whom four brothers and two sisters survive, the rest having died in infancy from causes unknown. Beyond having had measles in childhood, she enjoyed good health until lately. There is no history of personal injury. She began to menstruate at the age of thirteen; the flow generally lasted seven days and was excessive. Menstruation continued to be regular until two months ago, since which it has been absent.

Six months ago she noticed that she was getting paler, that she had almost constant headache, that she was easily tired after walking, and that she had palpitation and breathlessness on the least exertion.

Four and a half months ago she observed that her face and the backs of her hands were becoming yellow. They were so yellow that the case was at first regarded by her doctor as one of jaundice. Since then the color has become darker, and has spread to other parts of her body. At this stage vomiting occurred, frequently as often as three or four times a day. Since then she has occasionally felt sick but has not vomited. There has been steady emaciation, the palpitation has increased, and she now complains of vertigo when she attempts to walk.

She still remains moderately well nourished. Her temperature varies from 97° to 98° F. Her face is irregularly pigmented; it is of a dark-brown color,—darker in some places than others, especially the bridge of the nose, temples, angles of the mouth, and upper lip. There are dark patches, too, on the cheeks. The surface of her body, gener-

ally speaking, looks "freckled." The ears and the skin of the neck are pigmented. The skin of the scalp is pale, but at the border of the hair there is a sharp line of demarcation between the pale scalp and the pigmented brow. Where the two lips come in contact there are brown patches. Irregular dark patches are noticed inside the lips and cheeks, but there is nothing on the gums or tongue; although the hard and soft palate and under surface of the tongue exhibit well-marked patches and striæ of pigmentation.

There is also deep pigmentation along the free edges of the eyelids. The anterior folds of the axillæ are very dark, and there are dark patches irregularly distributed over the shoulders, the backs of the hands, and both aspects of the forearms.

The nipples and their areolæ are very black, and there is a belt of pigment round the waist above the crests of the ilia.

Whilst the skin of the abdomen is, generally speaking, of a pale brown color, the umbilicus is almost black. The groins are deeply pigmented. The thighs, legs, and feet are much less bronzed than the forearms, neck, and trunk. Below the knees are two broad pigmented bands where the garters were worn, and the cicatrices of two old sores at the right knee are deeply bronzed. The vulva, and especially the labia minora, are very dark—almost black; the vaginal mucous membrane is not pigmented. There is no evidence of spinal curvature, caries, or abscess. The lungs and heart are healthy; the pulse is feeble, easily compressed, and registers eighty-six beats per minute. The teeth are carious, and the gums here and there are ulcerated around their bases. The tongue is clean and moist. The abdomen presents nothing remarkable. The liver-dulness measures three inches. The splenic dulness is appreciable, and nothing abnormal can be detected over the kidneys. The urine varies from eighteen to forty-two ounces daily; it is acid, specific gravity 1018, no sugar, no albumen, no pus. Patient weighs six stone, five pounds (eighty-nine pounds). Treatment by calcium hypophosphite.

PROGRESS OF THE CASE.

July 7.—Vomiting returned a few days after her admission, and menstruation, which had been absent for two months, reappeared.

July 21.—A specimen of blood was sent to Professor Bedson. He found that whilst it gave the spectrum of hæmoglobin, it also contained some dark substance, the nature of which was unknown to him. The daily discharge of urea was 292.6 grains.

On the 22d of July twenty minims of supra-renal juice were in-

jected subcutaneously. The urinary flow gradually rose to eighty-two ounces, and the urea to 391.7 grains, daily.

July 25.—She says she feels better; is taking her food well; is allowed a moderately full diet,—meat, etc. Urine, sixty-two ounces; urea, three hundred and five grains daily.

Subcutaneous injections of supra-renal juice were made every four days. The temperature gradually rose to 98° and 99° F., and by the 10th of August her weight had risen to six stone, eight and a half pounds (ninety-two and a half pounds). On the 14th of August it struck all who saw her that the pigmentation of the face was less, so we continued the injections. From this to the 29th of September, the patient seemed to be progressing satisfactorily, but on this date it is noted that she is suffering from loss of appetite, great debility, and that there has been a return of epigastric pain and vomiting. Two days afterwards these latter symptoms had disappeared. It was the juice of the supra-renals of rabbits only that up to this stage had been employed; and as these organs are small and our results were not extremely satisfactory, we decided upon trying that obtained from the sheep. We were only able to administer two twenty-minim injections, for the patient's friends, tired with her long residence in the infirmary, took her home.

Looking back upon the case, I think I must admit that the subcutaneous injection of supra-renal juice seemed to moderate the pigmentation of the face,—generally speaking, it became paler,—but beyond this, and even with it, there was no striking result—no marked gain in strength and weight—that I could attribute to the treatment.

I saw the patient again on the 24th of February, 1894. It is more than four months since she left the infirmary. She says she is stronger, but I see her emaciated. The skin of the face is much darker, the terminal two-thirds of the fingers are dark bronzed, the hair is falling off, and the scalp is becoming brown.

Where the lips meet can be seen a broad line of pigmentation; inside the mouth the mucous membrane shows alternately dark and pale patches, and a dark line of pigment is noticed inside the cheek on a level with the upper border of the lower teeth and another just above it. The palate is studded with dark patches, and, while the upper surface of the tongue remains clear, a long pigmented line is seen running along the large right vein underneath that organ. The finger-nails are very dark, particularly at their origin, and on pressure the color does not disappear so readily as in health. Lungs and heart are healthy. Pulse one hundred, very small and compressible. (This rising pulse is of

interest, for as the disease progresses the tendency is for a paralytic tachycardia to develop, due, it is thought, to toxic material circulating in the blood and affecting either the myocardium or nerve-centres.) The skin of the trunk, generally speaking, is darker. She has not vomited since leaving the infirmary, but there is constant frontal headache. Urine shows just a trace of albumen, and contains six grains of urea to the ounce.

Blood, 2,550,000 cells in one cubic millimetre; the white corpuscles are increased, as many as forty being seen in the field; they are very granular; the red form rouleaux, and are pale.

In our patient are fully depicted all that is characteristic of Addison's disease. If I were to give you a picture of this disease, it would be a progressive asthenia often originating without any apparent cause, and seldom dating from any definite period; an indescribable aspect of listlessness or depression; great languor and indisposition for exertion; remarkable weakness of the heart's action; a small, feeble, compressible pulse; loss of appetite, irritability of the stomach, nausea, and retching.

These are symptoms almost invariably present. To these might be added pains in the loins, hypochondria or epigastrium, increased by pressure. In advanced cases frequent vomiting, breathlessness, and palpitation on muscular effort; occasional sighing or yawning; generally faintness or giddiness, particularly on rising or even on being raised in bed. With all these there may be little or no perceptible emaciation; the temperature, as a rule, is below the normal; the urine is often small in quantity, but not necessarily so, of low specific gravity and deficient in solids; the tongue is moist; the mind remains clear to the last, but in the latest stages of the disease the patient lies in a drowsy semi-comatose condition, from which he can be aroused to answer in monosyllables slowly uttered. As this stage is reached, the temperature falls still further below the normal, and death comes by sheer asthenia, sometimes suddenly, as if in syncope, or it may be that as life draws to its end there is incoherence or delirium, sometimes low or muttering, sometimes loud and active, the patient ultimately passing into a state of coma, in which he dies.

It is the bronzing of the skin that attracts attention and settles, with the symptoms enumerated, the diagnosis; but even before the discoloration occurs many of the symptoms are present, such as the increasing debility, the frequent vomiting, and the pains in the abdomen. If to these is added a small, feeble, and rapid pulse, the diagnosis of morbus Addisonii is almost certain; although, I must admit, that in

the early stages of the disease I have not found the pulse so rapid as many writers would lead us to infer. Contrasted with the slightly-bronzed tint of the skin, certain parts are extremely black,—e.g., the axillæ, nipples, and areolæ, certain parts of the abdomen, groins, and the genitals. Whenever pressure has been for long applied, there the skin is deeply pigmented.

We regard discoloration of the skin as frequently associated with disease of the supra-renals, but bronzing of the skin may exist without these organs being affected. There is, for example, the dirty-brown color of the skin met with in the very poor, whose skin is seldom washed and, too, frequently overrun with vermin. There is the discoloration caused by pityriasis versicolor, a condition here mistaken for Addison's disease; the peculiar brown color of the skin in some cases of exophthalmic goitre; the coppery-colored eruptions of syphilis; the peculiar bronzing of the skin of mariners who have sailed much in the tropics and suffered from malaria. Phthisis, chronic, hepatic, and malignant disease may also be followed by bronzing of the skin.

As the bronzing of the skin is met with under many circumstances, we cannot regard it as specially indicative of Addison's disease and pointing to pathological changes in the supra-renals. The fact remains that the supra-renals may be involved in neighboring malignant growths, yet no bronzing of the skin occur. That has long been recognized, and yet, on the other hand, it is just as true that when the capsules are the seat of a low form of inflammation which destroys their tissue,—the inflammatory exudate becoming cheesy, as in tubercular disease,—we find that during life there had been the bronzed skin and the symptoms already enumerated. Their association is such that we have come to regard this form of inflammation of supra-renal capsules as the pathological substratum of Addison's disease. Round this point, however, has centred much discussion. It has been argued that the symptoms do not depend upon the destruction of the capsules, but upon the inflammatory exudate pressing upon the nerves in their immediate neighborhood. We don't know exactly what is the function of the supra-renal capsules, but McMunn's observations seem to me to throw considerable light upon this important question. (*British Medical Journal*, February, 1888.)

The wide distribution of the supra-renals in mammals, birds, amphibians, fishes, and reptiles, is clearly an indication that they perform some important function in the economy, and that they must not be regarded as simply the remains of foetal structures. They have a large blood-supply, and are intimately connected with the nervous system.

Beginning as small yellow masses of a granular nature in amphibians, they become larger as we ascend the animal scale, until we find them in man as two well-developed, though small, organs lying close upon the upper border of the kidneys. Like ordinary glands, they are composed of two parts,—a medullary, which has been developed from the epiblast and in which the nerve elements lie, and a cortical portion, a derivative from the mesoblast.

It is only recently that our knowledge of the function of the supra-renals has assumed definite form. Brown-Séquard removed the supra-renals from animals, but the result was fatal, probably owing to hemorrhage and the injury inflicted upon nerve-structures, especially the semilunar ganglia, although Brown-Séquard himself regarded death as due to the circulation in the blood of poisonous material which it is the function of the glands to remove. Tizzoni's experiments are more in harmony with our view of the relationship of the supra-renals and Addison's disease. Removal of the capsules was in his case followed by a very distinct bronzing of the skin and mucous membranes.

The employment of the spectroscope by McMunn has further enlightened us. Finding hæmochromogen in the cells of the liver, bile, and supra-renals, he naturally regarded it as a pigment about to be excreted, and that in all probability, whilst the hæmoglobin was its major source, the histo-hæmatins contributed their share. McMunn found histo-hæmatin in the cortical portion of the supra-renals. He therefore concluded that "in the adrenals a downward metamorphosis of worn-out pigments—hæmoglobin and the histo-hæmatins—is taking place, and the function of these organs is to pick out of the circulation these worn-out or effete coloring matters with their accompanying proteids."

If, then, the adrenals discharge this function, we ought to find evidence in the excretions of incompletely metabolised pigments, when these pigment-metabolising organs are unfitted by disease for the performance of their function. And that is exactly what I found. I have detected, by means of the spectroscope, in the urine of Addison's disease such a pigment, and which I named "uro-hæmatin."

Whatever view we take of Addison's disease, we cannot but admit that toxic material is circulating in the blood. Foa and Pellacanni found that the injection of aqueous and alcoholic extracts of supra-renals was followed by toxic effects. We naturally ask ourselves the question, therefore, is it the function of the glands in health to remove such materials from the blood, and to transform them into substances of a less harmful nature preparatory to their elimination from the economy?

Opposed to the view which attributes Addison's disease to a *morbid condition of the supra-renals* is another known as the *nervous theory*, which seeks to explain the disease by a lesion of the abdominal-sympathetic, particularly the solar plexus. Expounded by Jaccoud, it has been warmly supported by Alezais and Arnaud, who maintain that the special bronzing of the skin is wanting so long as the lesion is confined to the interior of the supra-renal gland, but that it appears as soon as the periphery of the gland becomes involved, owing to the morbid changes induced in the sympathetic ganglia contained in the fibrous covering of the capsules.

Admitting for the moment that disease of the supra-renals is of itself not sufficient to give rise to Addison's disease, and that it is necessary to have pathological changes in the peri-glandular sympathetic nerves, or an irritative lesion from compression of the solar plexus or semilunar ganglia, I have yet to be convinced that these lesions of the nervous system do not in some way or other derange the functions of the supra-renal capsules.

I cannot explain the relationship of such nerve-lesions with pigmentation of the skin. Raymond has advanced the theory that the human skin naturally contains feebly pigmentary elements subject to nervous influences, just as the chromatoblasts of certain animals are under the immediate direction of a special innervation. Addisonian pigmentation is by him regarded as a nutritive disturbance in the pigmentary apparatus of the skin, depending upon an irritative lesion of the peri-supra-renal or semilunar ganglia, or of the solar plexus. Chauffard (*Medical Week*, January 23, 1894) insists upon a double lesion for Addison's disease,—viz., total destruction of the supra-renals and irritation of the neighboring ganglia, and that caseous infiltration of the capsules is liable to produce both lesions.

The success which has followed the treatment of myxoedema by ingrafting the thyroid or the subcutaneous injection of thyroid juice, has revived fresh interest in the pathology of Addison's disease. Glands not only remove material from the blood, they contribute their own special product to it. When Abelous and Langlois (*Archive de Physiologie*, 1892) removed the supra-renals from a frog, the operation was invariably fatal. Where one capsule was alone removed the animal survived; and, if after destruction of both capsules a fragment of kidney with its corresponding adrenal was inserted under the skin, the animal's life was prolonged. Langlois ascertained in the case of a dog, that so long as one-sixth part by weight of the supra-renal is left intact, the animal does not succumb to the operation at all; a circumstance strongly

suggesting, though not proving, that many of the symptoms of Addison's disease and death of the animals operated upon are due to a toxæmia consequent upon the circulation in the blood of material that is removed normally by these glands.

It was, therefore, in the belief that in Addison's disease we have, on the one hand, the blood poisoned by toxic material dependent upon the retention of effete pigments and their imperfectly metabolised associated proteids, and, on the other, that there was withheld from the blood what is normally contributed to it by the supra-renals, that I tried the injection of adrenal juice. I persevered with the treatment for a time, and whilst I admit that to all of us the skin seemed to become lighter in color and the patient slightly gained in weight, there was no striking effect produced.

Chauffard, who has tried a similar line of treatment, has experienced similar results. In his case little or no benefit followed from the injections, except, perhaps, a slight gain in strength; though even that was by no means certain.

As I am still of the opinion that the symptomatology of Addison's disease—the languor, vomiting, vertigo, and tachycardia—depends upon a toxæmia, I am hopeful that an increasing knowledge of the functions of the supra-renal glands will yet place in our hands a reliable, safe, and successful line of treatment.

EXOPHTHALMIC GOITRE; SPLENIC-MYELO- GENIC LEUKÆMIA.

CLINICAL LECTURE DELIVERED AT THE COOK COUNTY HOSPITAL.

BY JAMES B. HERRICK, M.D.,

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GENTLEMEN,—I am privileged in being able to show you this morning two typical cases of the rarer diseases that you will meet with in private practice, and yet diseases that all of you will see sooner or later in the course of your professional careers. The first case is one of exophthalmic goitre, and the other is one of splenic leukæmia. I think from any seat in the amphitheatre that any one of you will be able to make a differential diagnosis between these two cases even upon inspection. I do not need to tell you that this patient to the right is the one who suffers from exophthalmic goitre. From any part of the room you can see the exophthalmos, and those of you who are nearer can see the enlargement of the thyroid gland, constituting the goitre. Let us take up this case first, and I will preface what I have to say concerning it with the remark that it is a typical case of exophthalmic goitre. It is a valuable case, therefore, to present to you, because it shows in a characteristic manner the classical features of the disease.

You know that there is a trio, in exophthalmic goitre, of what may be called classical symptoms. These three symptoms are exophthalmos, goitre, and cardiac irritability. You can all see in this patient the prominence of the eyeballs, constituting the exophthalmos. You can see the enlargement of the thyroid gland, constituting the goitre, and I can feel, even through the clothing, that there is a rapid, tumultuous, forcible action of the heart; so that we have here, even upon the most superficial examination, a diagnosis of exophthalmic goitre. Let us see if we can learn from the clinical history in what manner this disease commenced.

The patient is twenty-nine years of age, unmarried, has been ac-

customed to doing house-work, and is a native of Norway. She presents a family history that is negative, save that the father died of consumption. She has lived in this city for five years. Her menstrual functions began at seventeen years of age, and she was regular in this respect up to about five months ago, but for five months there has been no menstrual flow. Her previous illnesses were practically none, save the ordinary diseases of childhood. Last July—that is, about six months ago—the patient became very nervous. She had *la grippe* in July, and this nervousness followed a fright, the exact nature of which she refuses to reveal, received about that time. Two months later she noticed that the eyeballs were prominent, and one month later than that the goitre was first noticed. There was a gradual increase in the size of the thyroid gland. About Christmas-time she first noticed fluttering of the heart. All these symptoms have been gradually intensified. She is now very nervous, crying upon the slightest excitement. She is annoyed at times by a trembling of the body which she cannot control. At times she passes an excessive amount of urine, rising for this purpose often at night. The appetite is capricious. She has lost several pounds in weight, her former ruddy complexion has become pale and sallow, and she feels weak and is easily exhausted. That is as typical and classical a history of the manner of development of the symptoms in exophthalmic goitre as you will ever meet with.

The efficient cause in this case is the fright and severe illness from which she suffered in July. It is not an uncommon occurrence for the symptoms of exophthalmic goitre to develop within the course of twenty-four hours, or even within one hour after some great shock of the nervous system. I have the authority of an eminent physician in this city for the statement that he has seen an enormous goitre develop within five minutes in such a case as this. There are numerous instances in which goitres much larger than the one we have here have developed in the course of a few days. Oftentimes, however, the goitre does not develop until after other symptoms have been noted. In this case, the nervousness preceded the exophthalmos.

Exophthalmos, the prominence of the eyeballs, is presumably due to the fulness of the orbital blood-vessels. This pushes the eyeball forward. There is another reason, too, why the eyes are prominent, and that is, the upper lid has a tendency to become retracted. This retraction of the lid is sometimes shown, although I do not think you will see it well in this case, when the patient is directed to look down. The upper lid does not follow the movement of the eyeball as it should, so that as the eye is rolled downward a wide expanse of white sclerotic

is exposed. I will see if that is present in this case. No; here the upper lid seems to follow fairly well every movement of the eyes. There is a tendency to paresis of the ocular muscles, so that the movements of the eyeball are restricted, and, the orbital cavity being exceptionally full, motion is limited in every direction. This is best seen by having the patient attempt to converge the eyes upon some object. Ordinarily the eyeball will follow the finger as it is brought closer and closer to the nose until the patient has marked internal strabismus. In many of these cases the patient is unable to do that. This lack of the movement of the eyeballs in this manner is sometimes spoken of as the sign of *Möbius*. It is with a good deal of difficulty, you will notice, that she converges the eyes to a near point. *Exophthalmos* is present in the majority of cases; yet there are cases of *Graves's disease* in which the *exophthalmos* is very slight or totally lacking. The wild, frightened, staring expression of the eyes (*Stellwag's sign*) is not as marked as it was before the patient came to the hospital.

The second one of the symptoms—the goitre—may, as I have previously remarked, develop suddenly or gradually. The size of the thyroid may vary, being slightly enlarged, or being so large as to interfere by pressure with swallowing and breathing. Often there is a symmetrical enlargement of the gland, as in this case, the isthmus and either lobe being quite uniformly increased in size. At other times the enlargement is asymmetrical. I feel a perceptible thrill over the enlarged gland. The gland is quite soft; a systolic murmur can be plainly heard. In other cases, especially those of long standing, the goitre seems to have a larger amount of connective tissue, and is hard and firm.

The third one of the group of symptoms, which was present somewhat late in this case, is that of cardiac irritability. There is scarcely a case of *Graves's disease* in which the heart does not show irritability. The patient is usually conscious of palpitation. A slight exertion, excitement, or emotion will cause the heart to flutter, to beat violently and tumultuously. The physician often finds the pulse 130 to 140, while perhaps at another time it is but 80 or 90. Irregularity is not infrequently found in the heart's action. Murmurs are detected, and these are not alone due to valvular disease, but are probably hæmic in origin in this case. This patient's pulse has been running somewhat erratically. On admission the pulse was 140; the next day I find it recorded 102, 120, 124, and 112. Two days later it was as low as 100, the next day varying between 84 and 104, the day following between 88 and 108, and still the following day it was as low as 72. On the

20th of January I find it recorded 102, and so it has varied between 72 and 140, though no irregularity is reported. In this case there is a distinct systolic murmur. The heart is slightly enlarged. There is nothing, however, in the previous history of the case to indicate valvular disease. This murmur is heard not alone at the apex, but to the left of the sternum and in the second and third interspaces, where the murmur known as hæmic is often best heard. Cardiac irritability should be numbered as one of the early symptoms in many cases of exophthalmic goitre. We hear physicians speak of tachycardia (rapid heart action). Many of these cases are probably cases of commencing exophthalmic goitre. They are not recognized, and perhaps cannot be recognized thus early, because at the time of examination there is no exophthalmos, no goitre; but it should be looked upon as a suspicious circumstance when a patient has occasional attacks of tachycardia and of nervousness.

I might almost say that nervousness and tremor make a fourth classical symptom of this disease, for in almost every case there are tremor and nervousness. The tremor may be very fine; the muscle may vibrate eight, ten, or twelve times to the second, or there may be coarser tremblings, such as you notice in this patient. As I examined the eyes yesterday the patient trembled perceptibly, and she told me that the trembling had annoyed her a great deal. I have seen patients in whom the trembling was so marked that it was impossible for them to carry a glass of water to the lips without spilling the water; they trembled as violently as you have seen patients with multiple cerebro-spinal sclerosis tremble on attempting to perform a voluntary motion. In fact, the trembling may assume almost a choreic character. The patient has in her history a record of crying upon the slightest provocation. Many patients with exophthalmic goitre are extremely nervous, emotional, even hysterical, and we may have the hysterical phenomena to the extent of hysterical paralysis, hysterical amaurosis, and various other phenomena that are characteristic of this peculiar affection.

The genito-urinary tract is almost always deranged. In these cases, which occur so often in women, amenorrhœa is the rule. This patient, you remember, has not menstruated for five months. There is a cessation of the menstrual function, or, at least, an irregularity in its performance. So, too, there is not infrequently atrophy of the genitalia. The mammary gland will atrophy in cases of this kind. Patients afflicted with this disease usually suffer from polyuria. The physician will sometimes think that he has a case of diabetes, because the patient will tell him that she urinates so frequently and passes such a large

amount of water. In this case that symptom is present; at least, the patient assures me that she has been bothered with frequency of urination. The exact amount of urine passed in twenty-four hours has not yet been determined. The urine is of low specific gravity, acid, chemical and microscopic examination negative. How far this polyuria is dependent upon Graves's disease alone, and how far it depends upon the hysteria which complicates it, is not clearly settled. You know in cases of hysteria polyuria is a very frequent symptom. The skin of these patients usually appears somewhat pale because of the slight degree of anæmia, and it also is characteristic that the patient suffers from profuse and frequent sweating. This patient does not present that symptom. Direct inquiry fails to elicit the fact that she has suffered from day or night sweats, or from profuse sweating of any kind. The hair sometimes falls out. Alopecia is not present in this case. The appetite may remain good, but it is oftentimes poor, as in this case; and diarrhoea usually occurs at some time during the course of the disease. The temperature of exophthalmic goitre is not usually high. At some time during the course of the disease fever is generally observed. I find only two records showing the temperature above normal. On admission the temperature was 101° in the axilla, and the respiration was 48, quick, shallow; at another time the temperature was 99.8° . Rapid, shallow respiration with deficient chest expansion has been often noted. Patients with exophthalmic goitre usually appear much as does this patient, somewhat emaciated, somewhat pallid. They complain of weakness; they are oftentimes incapacitated for work.

Cures of the disease are recorded. There is no remedy, however, that can be looked upon as a specific. The disease is usually treated symptomatically. Rest seems to have a beneficial influence. Tonics and alteratives are given; a change of climate is advised. In some cases a change to a high altitude seems to benefit them, and yet, on the contrary, there are cases in which high altitude seems to have an opposite effect.

I have hurried over this case in order that I might within the hour show you a second case which is as typical and one of great interest. I refer to splenic leukæmia.

SPLenic LEUKÆMIA.

The next patient I show you is a Polish woman, thirty-eight years of age, married, housewife, admitted to the hospital on the 12th of December, 1893. She remained only six days, was discharged at her own request, and came back to the hospital a few days ago. The im-

perfect history, which we have been able to obtain through an interpreter, is as follows :

Her father is living, but nothing is known as to the condition of his health. Mother died shortly after the parturient period. She has two brothers living and healthy ; no sisters. She has had ten children, eight died when young, the living children are healthy. Four years ago, when she came to the United States, she says that her feet, legs, and abdomen were swollen, and that she had severe pains in the abdomen. The cause of this dropsical condition she does not know. There has not been any other illness.

Her present illness she dates from a previous pregnancy, one and a half years ago. She has continued to work up to four months ago. She cannot tell us when she first noticed the abdominal swelling. She thought it might be a second pregnancy. Her complaint is of pain in the left hypochondrium. Her appetite is good, though she is quite thirsty. She is weak, dyspnoic ; says she has grown pale and emaciated. Suffers some from dizziness.

I wish you to notice that in her history, aside from the complaint of pain in the left hypochondrium, there is absolutely no characteristic of any particular disease.

Many cases of leukæmia come on very insidiously. Unless there is a complaint of pain in the region of the spleen, as in this case, the patient is oftentimes unaware that he is ill, merely thinking that he is a little out of sorts, a little run down, that he needs a tonic, and perhaps consults a physician with a history merely of malaise, of becoming tired easily, of suffering a little from palpitation of the heart, from shortness of breath, from some digestive disturbance ; he knows, perhaps, that he has lost a little flesh and looks paler than he formerly did. But there is nothing in such a history as that characteristic of leukæmia. Such a patient might be suffering from a chronic form of intestinal or gastric disturbance, from diabetes, or from latent carcinoma. It is only the physical examination in leukæmia that enables us to make the diagnosis. In this patient's case we have the complaint of pain in the left hypochondrium, which directs our attention to that region of the body, and investigation shows us what the patient means when she tells us that she thought possibly she was pregnant.

As the patient lies upon the table, I find upon examination of the abdomen that springing from under the costal arch on the left side there is a hard, smooth mass that reaches to the median line, and slightly beyond in the lower portion of the abdomen. The lower edge can be felt as low as the symphysis pubis. I will outline it in your

presence with ink, so that you can see its dimensions. Notice, if you please, the two notches, one above, the other below, the umbilicus. These notches are quite characteristic of an enlarged spleen. The mass is not very tender; it is smooth; the abdominal wall seems to move freely over it as though there were no adhesions. Upon percussion you will notice that there is dulness. While on the right side I get a tympanitic note, the dulness extends higher, you perceive, than the normal splenic dulness should. There is no fluctuation. The apparent origin of the growth from beneath the left hypochondrium, its shape, notched border, would lead us to suspect the spleen, even though we did not have confirmatory evidence of this being enlargement of the spleen. What confirmatory evidence have we in such a case as this? With the pallor of the patient, anæmia, with the enlargement of the spleen, and with the signs of progressive weakness and emaciation, we should, of course, examine the blood to see whether or not we have to deal with a case of leukæmia.

In leukæmia we have a marked increase in the number of white blood-corpuscles. Normally there are found to every cubic millimetre five or six thousand white blood-globules. In cases of leukæmia we may find that number increased to fifty, one hundred, five hundred thousand, even to a million. The normal proportion, you remember, between the white and the red is about one to five hundred. In cases of leukæmia that proportion is violently disturbed, and we find one to fifty, one to twenty, and even one to one. The first blood count was made in December soon after her admission to the hospital. It showed the red corpuscles to be 1,500,000, the white 200,000, to the cubic millimetre; the relation of the white to the red about one to seven and a half. The blood count since her admission to the hospital the second time, after, I may state, she had been upon arsenic, shows 2,800,000 red blood-corpuscles, the relation of the white to the red being one to twenty. Here we have more nearly the normal relation. There has been not only an increase in the number of red corpuscles, but a diminution in the number of white corpuscles, probably due in great measure to the administration of arsenic. Whenever you can look through the microscope and find ten, twenty, or thirty white blood-corpuscles in the field (using a No. 7 objective (Leitz) and a No. 3 eye-piece), you may assume with almost absolute certainty that you have to deal with leukæmia. I might almost say that where there are more than 50,000 white blood-corpuscles to the cubic millimetre we have leukæmia, though there are recorded some cases of leucocytosis in which 80,000 white corpuscles have been found to the cubic millimetre, in which the proportion of

white to red was about one to thirty. Such a case, one of carcinoma of the kidney with metastatic involvement of the lymphatic glands, is recorded by Von Limbeck.

Have we any other means of determining leukæmia? Suppose we meet with a case in which instead of having 5000 we have 50,000 white corpuscles, how do we know whether that is a case of leukæmia or a case of leucocytosis? Now, by leucocytosis I mean an increase in the number of white blood-corpuscles that is transitory and due usually to inflammatory disease with an exudate, or to a malignant growth. You will find leucocytosis, for instance, in pleurisy with effusion; you will find it in purulent meningitis, but not in tubercular meningitis. You find it in erysipelas where there is an exudate into the skin. It is found particularly in pneumonia where you know there is a marked exudation. Leucocytosis is not usually found in febrile diseases where there is not an exudate. It is absent in typhoid, in scarlet fever, and in measles. So we have to make a differential diagnosis between leucocytosis and leukæmia, because both diseases are characterized by an increase in the number of white blood-corpuscles. This is done by an examination of stained specimens of blood.

I shall not have time to give you all the details of the technique of the staining of blood specimens, but I must say a word or two with regard to the different kinds of white corpuscles that are found in normal blood, and how the blood-findings differ in such a case as the one in hand from the normal. I may say in normal blood there are five kinds of white blood-corpuscles. Still more minute differences are noted by some and a more extended classification made. First, we have a *small lymphocyte*, which is about the size of a red blood-corpuscle, and which by Ehrlich's method of staining shows a large nucleus; in fact, the corpuscles seem to be entirely nucleus; there is scarcely any rim of protoplasm left. These corpuscles make up twenty per cent. of the normal white blood-corpuscles. Then we have what are known as the *polynuclear leucocytes*, and, for a reason which I will speak of later, *polynuclear neutrophiles*. These are larger than lymphocytes. They have a nucleus that is irregular in shape, oftentimes twisted upon itself. It is T-shaped, S-shaped, kidney-shaped, and sometimes split up into two or three nuclei. Not only do we find the nucleus of this shape decidedly irregular, but we notice that there is a distinct mass of protoplasm which contains granular matter, fine granules, which by the proper method of staining show that they stain with a neutral dye. I mean a dye that is not acid, not basic, but one that is a combination of the two. It is, therefore, a neutrophile. These make up say sixty-five

per cent. of the leucocytes. Now, between these two there are varieties that are known as *large mononuclear lymphocytes* and *transitional forms*, but I shall not take up much of your time in dwelling upon this phase of the subject. They are larger, and the nuclei seems to have a tendency to approach the polynuclear form. Within the protoplasm is seen an attempt at granulation. These together make up about eight per cent. These figures, however, are only approximate and vary in conditions of health. These are four varieties of corpuscles, and the fifth variety is the *eosinophile*. The eosinophile is large, has a distinct nucleus, its protoplasm contains granular matter, that resembles somewhat in its refractile qualities fat-globules; in fact, you sometimes think the leucocyte has fat in it. The granule has this peculiar quality. While the granules of the polynuclear neutrophiles stain with a neutral dye, those of the eosinophile stain with an acid dye. They stain, for instance, with eosin, which is an acid dye, and Ehrlich spoke of them as oxyphilic or eosinophilic from this property. Eosinophiles make up from two to five per cent. of the white corpuscles of normal blood. Thus we have from twenty to thirty of the small mononuclear forms, transitional forms from six to eight, polynuclear forms from sixty to eighty per cent., and the eosinophiles from one to three in every hundred of leucocytes.

What do we find in leucocytosis? The only mobile leucocyte is the polynuclear neutrophile. The only one that possesses amoeboid movements is the polynuclear neutrophile, which is the leucocyte that is found in exudation. *It is the one that is increased in leucocytosis.* If I examine the blood of leucocytosis, I may count the white blood-corpuscles and find the proportion, instead of being one white to five hundred red, one white to seventy-five red. There is a marked increase in the number of white corpuscles that might lead me to regard the case as leukæmia. But if I stain the specimen and differentiate between these varieties of leucocytes, make a differential count of a thousand of them, I find that the increase is solely in the polynuclear neutrophiles. This excludes leukæmia and establishes a diagnosis of leucocytosis. I have seen a case of leucocytosis, in Hodgkin's disease, where ninety-five per cent. of all the leucocytes were polynuclear neutrophiles. This is a striking change from the condition of the normal blood, and was the only means of distinguishing, in the case I refer to, between Hodgkin's disease and leukæmia.

What would we have in a case of leukæmia?

If we examined the blood we should find that the lymphocytes are not increased; we should find the transitional forms are not markedly

increased; we should find that the polynuclear neutrophiles are not increased, and I will add that the eosinophiles are not ordinarily increased, but there is a new white corpuscle that has come into view, which has a large solitary nucleus. It is of larger size than any yet described, and has neutrophilic granules without it. It is a large mononuclear neutrophile and is a pathological white blood-corpuscle. It is the myelocyte. It is supposed to have its origin in the marrow of the bones. It may be present in varying proportions, say in twenty-five out of every hundred. We should find also in splenic leukæmia that the eosinophiles are absolutely increased; relatively they are not always increased, though they may be. We might find, instead of two to four per cent., that in this case we should have four, six, or even eight per cent. of eosinophiles. The eosinophilous cell in the stained specimen not only looks beautiful under the microscope, but makes a striking picture. When there is even an absolute decrease in the eosinophilous cells, on looking through the microscope, this cell is the first thing to attract attention, so that following Ehrlich's early teaching it has often been erroneously stated that an increased number of eosinophiles was the most characteristic blood change in leukæmia, and was diagnostic of that disease. That is not so. I have found in ordinary asthma thirty-three per cent. of all the white blood-corpuscles eosinophiles. They have been found also in measles and other conditions. We find that the polynuclear neutrophiles have become relatively diminished. Instead of being sixty-five per cent., we might find but fifty. The increase then is particularly in the pathological white blood-corpuscle,—the myelocyte.

In the case of this patient that very thing is found. There is found a marked increase in the white corpuscles as shown by the blood-counting apparatus. There are found in abundance large mononuclear leucocytes with neutrophilic granules in the protoplasm. These are myelocytes, and are diagnostic, so far as we know, of spleno-myelogenic leukæmia. If this were a case of lymphatic leukæmia, we would have the lymphatic glands enlarged, and the disease probably pursuing a much more acute course. We should have something entirely different in the blood. There would be no myelocytes, but an enormous increase in the small lymphocytes. I have not seen a case of this kind, but I can give you the figures from Thayer, who records a case where he found in all the white blood-corpuscles ninety-seven per cent. of small mononuclear leucocytes. That presents a wonderfully different picture under the microscope from that presented by normal blood or by splenic-myelogenic blood.

The following table will enable you to see at a glance what a striking difference there is in the blood-findings in normal blood, leucocytosis, splenic-myelogenic leukæmia, and lymphatic leukæmia :

	Normal blood.	Leucocytosis.	Splenic-Myelogenic Leukæmia.	Lymphatic Leukæmia (Thayer).
Lymphocytes	15-30	4	1	97.9
Large mononuclear leucocytes and transitional forms	5	5	6	.4
Polynuclear neutrophiles	75	90	65	1.4
Eosinophiles	2-5	1	5	.1
Myelocytes	0	0	23	0.0
Total	100	100	100	100

There are certain other things found in the blood that should be mentioned. Nucleated red corpuscles are found in leukæmia as in pernicious anæmia. They are present in this case. The marrow of the bones is oftentimes involved in these cases. Let us see if we can by pressure elicit any pain which sometimes shows an involvement of the bones. There seems to be no pain over the lower or upper extremity. The cranial bones do not seem to respond to pressure. The sternum is the bone that shows it oftener than any other. I find one spot in the sternum in its lowest third that is very tender. Probably the bone marrow is oftener involved in these cases than can be proved clinically.

I might say with regard to the other symptoms that they are practically those of anæmia,—loss of strength, shortness of breath, palpitation of the heart, dizziness, tinnitus, and often a tendency to hemorrhage. Nose-bleed is common ; sometimes vomiting of blood will cause sudden death. Hemorrhage from the bowels and kidneys is noted. As the patient becomes weaker and more and more anæmic, œdema is apt to appear in different parts of the body. In this case it is not present. The pleural, pericardial, and peritoneal sacs are free from fluid.

As to the cause of the disease in this case, we have very little to help us. I think we are warranted in suspecting syphilis, but it is only a suspicion. I find suspicious scars in two or three places,—one on the patient's back, one on the leg, and a few small ones on the face. She lost eight children in infancy. That of itself would point to syphilis. This disease is often looked upon as a cause of leukæmia. Malaria is frequently assigned as a cause of this disease, and certainly many patients with leukæmia have been sufferers from previous malarial infection. Of five cases of splenic-myelogenic leukæmia that I have seen in the past



FIG. 1.—Immediately after the introduction of the needle.



FIG. 2.—Showing the blood flowing from the canula.

twelve months, four have been in women. This is contrary to the facts of ordinary statistics. Males are oftener affected.

There are a great many points of interest in connection with this disease, especially with the blood-findings, that we could take up if there were time to do so. Remember, if you cannot diagnose a case of leukæmia upon the symptoms alone, upon the finding of an enlarged spleen or enlarged lymphatic glands, you have to make an examination of the blood. In cases where you find the white blood-corpuscles enormously increased you can make a diagnosis without staining the blood. Moreover, you can make a differential diagnosis between the varieties, because if you find the field studded with *small* white blood-corpuscles, even though you do not stain them, you may say they are lymphocytes, with their origin in the lymph glands. Virchow made that distinction before methods of blood-staining were in vogue. If you find an increase in *large* leucocytes, you may assume you have a case of splenic leukæmia, or the complicated variety, splenic-myelogenous leukæmia. In cases where you cannot tell from the symptoms, nor from the examination of the unstained blood, whether you are dealing with a case of leukæmia or leucocytosis, you have to stain the blood, and the method of staining I can perhaps take up on some future occasion in this clinic.

So far as the treatment of these cases goes, it is rare that a cure is effected. There are probably no drugs that exercise a better influence than tonics and the alteratives, and particularly arsenic. Arsenic will cause a reduction in the number of white blood-corpuscles. You may be misled sometimes in your diagnosis of such cases where arsenic has been used. We will suppose that the patient has been under arsenic treatment for two months; she then goes out of the hospital, and in two or three days gets into the hands of a physician. He finds an enlarged spleen, and, suspecting leukæmia, he examines the blood and is surprised to find that the proportion of white to red blood-corpuscles is practically normal. He may not know that arsenic has caused that reduction in the number of white blood-corpuscles; consequently he will make a mistake in diagnosis unless he stains the blood. If he stains the blood he will find myelocytes, even though they are few in number. He will find that there will be pathological corpuscles.

I trust that this case will impress upon you the fact that there is a great deal of value in staining the blood, and that it is especially valuable for the differentiation between the varieties of white blood-corpuscles, by which differentiation we may recognize readily cases of leucocytosis and the varieties of leukæmia.

HÆMATOLOGICAL INVESTIGATIONS IN SEPSIS AND ALLIED CONDITIONS.

CLINICAL LECTURE DELIVERED AT THE CHARITÉ HOSPITAL.

BY DR. E. GRAWITZ,

Privatdocent and Assistant in the Second Medical Clinic of the University of Berlin.

GENTLEMEN,—In reviewing the relatively rich literature on the subject of septic and allied conditions, we find that the changes which the individual constituents of the blood suffer in these affections are described in variously different ways, and that most authors on this subject, among whom I may name Manassein, Quincke, Heyl, Maisurianz, Mobitz, Von Götschel, Halla, Bond, Tumas, Maragliano, Kanthak, Rieder and Von Limbeck, confine themselves principally to descriptions of the changes which the corpuscular elements undergo, disregarding almost wholly the chemical constitution of the blood proper. In one point only these authors seem to agree, and this is that in septic processes the blood becomes poorer in its solid elements, while even the occurrence of a leucocytosis, though quite generally considered invariably present, has in a few cases not been met with.

Because of these diverging views it seemed desirable, in order to obtain a clear picture of all the important blood changes in septic diseases, to institute numerous investigations, directed in as many different lines as possible. I have found rich material for the study of sepsis, and more particularly puerperal sepsis, in the clinic of Professor Gerhardt, and have also studied several cases of malignant endocarditis. Before detailing the points elicited by these experiments I think it a matter of the greatest importance to lay stress on the method of withdrawing blood from the living subject, for this is a requirement that must be properly carried out in every investigation of blood, the results obtained in the abstraction of blood by means of leeches differing essentially from those obtained by simple incision or venesection. In the case of leeches by the process of suction the blood is diluted by the admixture of an undeterminable amount of lymph withdrawn from the

tissue; and the blood that is obtained by a superficial incision, which also necessarily opens numerous lymph spaces, is thereby made thinner than that which is taken directly from a vein.

This last method, the abstraction of blood from a vein, I have applied methodically in all my investigations, by first lightly compressing the superficial veins of the upper arm, then introducing a sharp, medium-size canula into the vein selected, directly through the skin. The velocity of the flow of blood is proportionate to the size of the canula. (*Vide* photographs.)

The whole method is, of course, carried on under thorough anti-septic precautions, the site of puncture being carefully disinfected and the metal canula sterilized by direct heat. This method of abstracting blood has the advantages of easy execution, of causing no greater discomfort to the patient than the pricking of a needle would, and of being without danger; and, furthermore, the blood can be allowed to trickle in any desirable quantity (several grammes) into conveniently placed graduated vessels, without danger of admixture of tissue lymph, the rapidity of the whole process guarding against any alteration of the blood as a result of dissipation of blood gases during the abstraction. I have never encountered an accident or secondary complication in the application of this method. The following points were determined in the venous blood abstracted in all cases in the manner described above:

1. The amount of dry residue. I might remark here that by far the greater quantity of dry residue is formed from the albumin of the blood, so that the determination of the dry residue gives us a very safe means of estimating the albumin in the blood. I have found the *average amount of dry residue* from the blood of numerous healthy people to be 21.5 per cent. These results were obtained by catching one or two grammes of blood in small scale-pans, hermetically sealing these and then subjecting them to the process of desiccation over sulphuric acid in a vacuum. This method gives the most satisfactory opportunity of obtaining accurate results.

2. The dry residue of the blood-serum was ascertained by the same method. For this purpose two or three grammes of blood were caught in a small scale-pan, allowed to coagulate in a cool spot, and the serum removed, with a capillary pipette, after a lapse of twenty-four hours. The residue was then estimated as described above. *The average amount of residue from the blood-serum of healthy individuals was found to be 10.75 per cent.* It is advisable to previously sterilize the receptacles used for the preparation of blood-serum in order to prevent the development of bacteria of decomposition.

3. The number of red corpuscles was estimated with the Thoma-Zeiss hæmocytometer, and,—

4. The white corpuscles determined in the usual manner.

5. The morphological changes of the red corpuscles were studied with reference to their changes in form (poikilocytes, microcytes, and macrocytes), and the white corpuscles, according to their nucleus formation and granulation of the protoplasm (neutrophil and eosinophil).

6. In order to demonstrate the possible existence of bacteria in the blood I made cultures in a number of cases *on several nourishing media*, and for this purpose in particular can highly recommend the previously described method of abstracting the venous blood by puncture.

It is hardly likely that we would expect to find, in the case of a bacterial affection, the bacteria in such large numbers in the blood as to obtain successful cultures from every drop drawn. On the contrary, I have satisfied myself by numerous experiments, that it is necessary to inoculate larger quantities of blood, and that by so doing one has the best opportunity of developing bacteria in doubtful cases. It is therefore a feasible plan to hold test-tubes containing bouillon under the end of the aseptic canula when in position, and thus allow the blood, safe from contact with instruments or other contamination, to trickle into the bouillon in any desired amount. By this means we can make a culture of the blood with the least possible chance of contamination, and, furthermore, the larger the quantity of blood used the more positive the results. In like manner the blood may be allowed to drop upon agar that has been allowed to harden in inclined tubes, upon blood-serum or other culture media.

Acting in accordance with the principles above mentioned, the experiments with cases mainly of septic-pyæmic fevers and malignant, ulcerative endocarditis gave the following results, which are detailed in the order of the previously described factors in the examination of the blood :

The dry residue of the blood I found diminished in all cases,—in those of little severity to seventeen to nineteen per cent., in those of moderate severity to thirteen to sixteen per cent., and in some of the very severe cases the reduction reached 11.33 per cent., 11.13 per cent., and 10.58 per cent. This reduction in the dry residue of the blood continued steadily and in proportion to the duration of the fever, and, in fact, in some cases the reduction was so rapid in its progress that I was able to demonstrate a daily loss of almost one per cent., an estimate likewise of the enormously rapid diminution of the blood-albumin. In the case in which the residue is mentioned as having been

10.58 per cent. the blood itself had a consistency considerably thinner than normal blood-serum.

From the above one can see how great is the injury sustained by the blood in septic processes. This enormous loss of albumin is apparently not incompatible with life, for I have seen quite a prolonged existence in several patients, the residue of whose blood had reached a very low figure. It was noticed, however, that all patients suffering from septic fevers and in whom the blood residue had fallen below fourteen per cent. died, so that it appears possible that these estimates of the concentration of the blood may not be without some prognostic value. For the sake of comparison I have made numerous experiments with other acute infectious diseases, especially with typhoid fever, pneumonia, etc., in which the fever reaches a much higher degree than in the septic cases mentioned above, but in no case have I been able to discover so rapid a reduction of the albumin in the blood.

The dry residue of blood-serum is also diminished, and, as in the case of the blood proper, is likewise proportionate to the duration of the illness. There seems to be, however, a definite, as yet unexplained, relation between this blood-serum residue and the degree of fever. This residue may fall rapidly in illnesses of sudden onset, though it may reach a comparatively higher estimate in the course of same, or even shortly before death; this may be explained as due to a transudation of serum from the blood into the tissues. In general, however, the serum residue was also much more markedly diminished in the septic fevers than in the acute infectious diseases, and I have estimated in the severe cases as low as seven per cent., 6.26 per cent., and 6.17 per cent.

If these two values—viz., that of the dry residue of the blood proper and that of its serum—are compared, one can see that the large reduction of albumin of the blood is due, in part, to the very considerable thinning of the serum; and, furthermore, that in consequence of this the hæmaglobin of the blood is not reduced to the degree that at first sight seems probable. The result of the examination of the red corpuscles in individual cases concurs with these findings.

The number of red corpuscles has been found materially diminished in all cases, averaging two or three millions per cubic millimetre in cases of moderate severity, and in a few cases of great severity reaching 1.8 to 1.4 millions. If, therefore, we compare the number of red corpuscles with the residue of the blood as measured by the value found for the serum, it seems that both values, including in the former the amount of hæmoglobin, are correspondingly reduced, and that therefore the individual red cells suffer no material loss of hæmoglobin.

These results are contrary to those found by Heyl,¹ Maissurians,² Mobitz,³ and Von Götschel⁴ in the clinic at Dorpat, who claim that in the septic infections of animals in particular there is a frequent difference between the hæmoglobin and stroma contents of the corpuscles.

We are now confronted with the question as to the explanation of this deterioration of the blood proper, whether it be rapid in its onset or slowly progressive, because other acute infectious fevers, as typhoid, in which the temperature reaches a much higher degree, do not approximate the low blood-values of septic fevers of even short duration.

The first theory, and it is one of considerable plausibility, is that, as a result of the presence of toxic substances that gain entrance into the blood from septic foci, the corpuscles are destroyed and the blood proper vitiated in consequence. I believe, in fact, that, more especially in the very severe cases, this destruction plays an important part, and I find this opinion strengthened by an observation of a case of very acute sepsis, which I think of sufficient interest to report in greater detail.

A woman forty-four years old was admitted to the clinic on September 15 with the history—which her relatives had to give for her—of being in the second or third month of pregnancy, and in good health until the previous day. On the night of this day, at eleven o'clock, severe uterine hemorrhages occurred, the contents of the womb passing away. Later developments proved that a midwife had attempted various manipulations for the evacuation of the uterine contents. During this very night the husband noticed blue spots appearing in his wife's face, and immediately brought her to the Charité.

The patient presented a peculiar appearance: her skin was of a dirty-yellow hue, the conjunctivæ of a deeper yellow, the face bronzed, and the nose and cheek deeply cyanotic, the face as a whole offering such a withered appearance as to suggest at first sight an attack of cholera, though all parts of the body had high temperature. The sensorium was cloudy, temperature 40° C., pulse 160, small and rapid; respirations 40 per minute, but regular. Thoracic viscera were normal.

The *urine*, which it was found necessary to draw by catheter, was

¹ Heyl, Zählungsergebnisse d. farblos. u. rothen Blutkörperchen. Dissert. Dorpat, 1891.

² Maissurians, Experiment. Studien über die quantitativen Veränderungen der rothen Blutkörper im Fieber. Ibid., 1882.

³ Mobitz, Experiment. Studien über die Veränderungen des Hämoglobins in Blute bei septischen Fieber. Ibid., 1883.

⁴ Von Götschel. Ibid., 1883.

dark red in color and thick in consistency, and contained free particles of hæmoglobin, but no red corpuscles.

The patient vomited bloody, mucous lumps, the examination of which for possible poisons proved negative.

The gynæcological examination discovered a wide-mouthed cervix, which easily permitted the introduction of two fingers into the uterus, which in size approximated a chicken's egg, and contained a number of odorless coagula.

The bacteriological examination of these odorless blood-clots proved of special importance for the diagnosis of this singular disease, for a smear culture of these coagula discovered them to be saturated with enormous masses of minute cocci, which proved, in a culture made later, to be composed largely of pyogenic streptococci; and, furthermore, it proved the danger of judging the non-infectious nature of abortion masses by their favorable external appearance or absence of bad odor.

The diagnosis of foudroyante sepsis from an infected uterus was made, and, death having followed a few hours after admission, was verified at the autopsy demanded by the court. Under these circumstances the examination of the blood (venous) was of special interest. The blood-count showed three hundred thousand red corpuscles per cubic millimetre, and a scant number of white corpuscles; the blood proper yielded a residue of 14.5 per cent. and the serum 13.1 per cent. This high value of the serum is to be attributed to the fact that it had a red color from the large quantity of hæmoglobin which it held in solution, as was easily proved by the guaiac-turpentine test.

We have here, therefore, a very acute case of a constitutional septic infection, which resulted in a hæmatolysis of intense degree, in consequence of which more than ninety per cent. of all the red corpuscles were speedily disintegrated, as was demonstrated by the blood-serum and urine, while at the same time the existing icterus pointed to an increased determination of the broken-down red corpuscles to the liver. I think that the importance of these observations justifies this rather detailed account of the case.

Although we find in this case direct evidence of a disintegration of the red corpuscles, I must say, on the other hand, that in no other case was such a disintegration demonstrable, for this would have brought about a hæmoglobinæmia, in a less degree, to be sure, than in the case cited; repeated efforts, even spectroscopic examinations, failed to reveal traces of hæmoglobin in the clear decanted serum. Whether any special organ, the liver in particular, becomes the seat of an exaggerated de-

struction of red corpuscles in the course of one of these septic fevers, I care not to say, though this destruction can under no circumstances be a very pronounced one, because if such were the case we would certainly find increased biliary secretion and an increased excretion of hydrobilirubin into the urine.

I believe that this degeneration of the blood may to some extent be explained by certain observations which I communicated on a previous occasion.¹ I found while studying the effect of different bacteria on the blood proper, that cultures of bacteria—for instance, cholera and diphtheria—have a tendency to thicken the consistency of the blood, for the injection of the cultures into the circulation is followed by a transudation of fluid from the blood into the tissues, in a way similar to that described by Heidenhain in his well-known work, “*Über Lymph-treibende mittel*,” having reference to cancer-juice and other substances. I demonstrated further, that, on the contrary, the injection of cultures of pyogenic cocci (*staphylococcus pyogenes aureus*, *streptococcus pyogenes albus*, etc.) gives the blood a thinner consistency by abstracting fluid from the tissues, this fluid entering the blood and diluting it. Since it was discovered at the same time that the cause of this transudation of lymph and consequent thinning of the blood, as mentioned in the last case, was due more to the substances elaborated by the bacteria than by the organisms themselves, I find in this a suggestion as to the cause of the thinning of the blood, so constantly observed in those suffering from septic diseases. This supposition seems even more likely, since it is the blood-serum in particular that is affected in these cases, and to a considerable degree.

As a rule, the red corpuscles show no distinct morphological changes in sepsis. In the very severe cases only do we find irregular forms, such as microcytes, macrocytes, and poikilocytes. Nucleated red corpuscles are very scantily present.

Much attention has for a long time been given by individual authors to the consideration of the number of white corpuscles in these cases. Among the more recent of the numerous observations that have been made, I may cite those of Rieder,² who found a considerable degree of leucocytosis in several cases of sepsis, and observed in one case, with temperature at normal, a total of thirty-seven thousand five hundred

¹ *Zeitschrift für Klinische Medizin*, 1893, E. Grawitz. “*Klinisch. Experimentelle Blutuntersuchungen*.”—Teil II.

² Rieder, *Beiträge zur Kenntniss der Leucocytose und verwandten Zustände des Blutes*. Veröffentl. aus d. med. Klinik in München, 1892.

white corpuscles. He came to the conclusion, therefore, that in almost all cases of sepsis—even those without exudation—a leucocytosis was demonstrable.

Von Limbeck,¹ on the contrary, was unable to demonstrate a leucocytosis in puerperal sepsis, and found in this a confirmation of his views, that in those infectious diseases only in which there existed an exudation into the tissues, an appreciable increase of white corpuscles during the fever period was to be found. Furthermore, he showed by experiments that the pus-producing staphylococci and streptococci in particular are such as excite leucocytosis in lower animals.

Maragliano² demonstrated that in acute infectious diseases there existed no connection between the severity of the case and the degree of leucocytosis, as had been claimed by some.

My own observations were in accord with those of most authors in that almost all cases showed a considerable leucocytosis, and in two cases only did the white corpuscles approximate the normal. In my cases, too, there was no relation between the degree of leucocytosis and the severity of the disease, nor did any fixed relation exist to the number of red corpuscles or residue of blood and serum. In other respects, however, some interesting results, as regards the presence of leucocytosis, were found in several cases. In two cases of puerperal sepsis the white corpuscles were in extraordinary abundance as a result of complications,—pneumonia in one case and circumscribed pulmonary gangrene in the other. Moreover, those septic pyæmic cases in which there was a localization of the process and those in which there existed an exudation into the tissues were attended with a more marked increase of the leucocytes than was the case in simple, uncomplicated septic states.

The white corpuscles themselves belonged largely to the variety of polynuclear cells with neutrophil granules. It was, however, impossible to demonstrate an increase of lymphocytes or eosinophil cells.

The bacteriology of these cases is of special interest. In but one case of simple puerperal sepsis was the presence of bacteria in the blood demonstrable,—the staphylococcus pyogenes aureus was found and a pure culture grown on suitable media.

My results were better and more positive with endocarditis ulcero-rosa. The first case was that of a seamstress twenty-eight years old, of

¹ Von Limbeck, *Klinisches und Experimentelles über die entzündliche Leucocytose*. Zeitsch. f. Heilkunde, Bd. x.

² Maragliano, *Beiträge zur Pathologie des Blutes*. Verhandlungen des XI. Congresses f. Innere Medicin, 1892.

medium height, with poorly-developed and flabby muscles and very little fat. Towards the end of October, 1893, the patient became ill, complaining of swelling of both ankles and severe vomiting, occasionally bloody. There was also an irritative cough with little expectoration. The patient became emaciated to an extreme degree in three weeks, and entered the Charité about the middle of November. The boundaries of the heart were, fourth rib above, left sternal border on the right, and mamillary line on the left. Loud systolic murmurs were heard over all the valves and a diastolic murmur over the apex, though otherwise the second sounds were clear; bronchitic murmurs were heard over the lungs.

The cervical, axillary, and inguinal glands were swollen, the abdomen much distended with gases, and distinct fluctuations existed in the dependent portion.

The patient, who was excessively anæmic, was examined on November 15 at a temperature of 38.2° C., and the following blood condition found:

Number of red corpuscles	2,750,000
Number of white corpuscles	8,800
Proportion of white to red cells	1:812
Dry residue of the blood	11.33 per cent.
Dry residue of the serum	7.02 "

The examination on November 16, at a temperature of 36.8° C., gave:

Dry residue of blood	10.58 per cent.
Dry residue of serum	6.84 "

Cultures made at the same time on bouillon and agar that had congealed in an inclined tube showed in two days delicate punctiform glass-gray colonies, which proved to be streptococci, and caused a violent phlegmonous swelling when injected into the back of a guinea-pig. We found thus a proof of the presence of streptococcus pyogenes aureus in the blood, and upon this, in connection with the heart-lesions, was based the diagnosis of endocarditis ulcerosa.

Death resulted on November 18. The pathological report was as follows: Anæmia universalis, *endocarditis aortæ et mitralis chronica retrahens, ulcerosa et verrucosa recens*, dilatatio et hypertrophia cordis, metamorphosis adiposa myocardii, myocarditis, hydrops universalis.

Small particles were cut from the ulcerated areas of the valves and smear cultures and sections made from these. Both showed the same streptococci that were found in the blood-cultures intra vitam; the out-

ermost strata of endocardium were found in the sections to be swollen and covered with thick masses of the above-mentioned bacteria, which could be followed into the deeper layers of the endocardium in the shape of fine lines. The bacteriological examination thus demonstrated *intra vitam* the probability of the existence of a malignant valvular process in this case, and this was thoroughly corroborated by the post-mortem finding.

The following case is of special interest because of the rare bacteriological condition therein found.

August W., thirty-two years of age, a laborer, consulted a physician in 1881 for a rushing of blood to the head and for indisposition, and was told that he had heart-disease. Before this period the patient had had no symptoms of the disease, but since then he suffered occasionally from palpitation and a rush of blood to the head, and quite frequently noticed painless blue spots on his body. On Whitsuntide, 1893, he had an attack of epistaxis following a number of severe attacks of congestion. This epistaxis lasted four days and nights and was finally controlled by nasal tamponing. This state of affairs continued during the summer: Occasional attacks of epistaxis, during which the patient felt ill and unable to work, and daily attacks of fever with profuse sweats and chills, were complained of. In the early part of November he was taken with pains in the calf of his right leg, his shoulders, and back, and with this a return of the epistaxis.

He sought relief at the Charité on November 7. All over his body, especially on the upper extremities, were found petechiæ.

Posteriorly on the right side, and extending down from a two-finger's-breadth distance below the angle of the scapula, there was quite a well-marked dulness. Above this line the breath-sounds were weak and attended with numerous râles. Auscultation and percussion showed normal lung substance in all other areas. *Fränkel's diplococci* were found in the rusty sputum. There was visible pulsation in the supra- and infra-clavicular fossæ in the region of the sterno-cleido mastoid, the facial artery, and the superficial temporal. Friction of the skin brought out a capillary pulsation.

The cardiac area was extended two finger-breadths' distance to the left of the nipple; the pulsation was distinctly visible in the mammary line in the fifth interspace, and a systolic thrill could be felt on palpation. Over all the valves a systolic murmur could be elicited, and a diastolic in addition at the aortic area.

The spleen was distinctly enlarged, and could be palpated under the border of the ribs.

The lower border of the liver was three centimetres below the border of the ribs in the mamillary line, and in the median line of the body two centimetres above the navel.

The urine contained albumen.

The temperatures were: November 18, morning, 40°, evening, 39.5°. November 19, morning, 37.5°; evening, 36°.

The blood examination made on November 8, at a temperature of 39.2° C., gave the following result:

Number of red corpuscles	4,400,000
Number of white corpuscles	16,800
Relation of white to red cells	1:262
Dry residue of blood	16.48 per cent.
Dry residue of serum	9.21 "

The blood examination made on November 9, at a temperature of 38.6° C., was as follows:

Number of red corpuscles	4,000,000
Number of white corpuscles	12,000
Dry residue of blood	15.78 per cent.
Dry residue of serum	9.88 "

The bacteriological examination of the blood made upon agar that had been allowed to solidify in inclined tubes showed a comparatively large number of small, delicate, grayish-white, disseminated colonies. These were found made up of diplococci that seemed smaller and rounder than the usual forms of the diplococcus pneumoniae of Fränkel, and grew nicely and without contamination in all cultures. Animals—white mice—into whose peritoneal cavities masses of these cocci were injected, died in twenty-four hours, and examination of their blood, and especially of the enlarged spleens, revealed numerous masses of encapsulated lance-shaped diplococci; in short, typical pictures of the diplococcus of septicæmia, corresponding to the diplococcus of Fränkel. We were, therefore, dealing with this bacterium, which, as is always the case in the agar culture, grew unencapsulated and presented a slightly changed form.

The whole clinical picture pointed to a malignant, ulcerating endocarditis, localized more particularly on the aortic leaflets; and, furthermore, the bacteriological finding justified the assumption that this ulcerative process was due to the diplococcus of Fränkel.

In this case, also, the rapid reduction of blood-residue, amounting to one per cent. in twenty-four hours, is worthy of note.

Death took place on November 10. The pathological examination

gave the following result: *Endocarditis aortica ulcerosa*, aneurysma valvulæ aorticæ ruptum, endocarditis mitralis verrucosa recens. *Pneumonia fibrinosa duplex*. Hyperplasia pulpæ lienis. Cicatrices et infarctus venum. Atrophia cyanotica hepatis.

In this case, too, small particles were taken from the ulcerated aorta and cultures made from smear preparations; sections were also made, and in all cases pure uncontaminated diplococci pneumoniæ of Fränkel were found. These were likewise found in encapsulated form upon the endocardium, and, as in the previous case reported, the outermost layers of the endocardium were more particularly involved in bacterial accumulations, only a few nests being found in the deeper structure. The Gram stain was used with much success.

In this case, too, the clinical examination and the bacteriological investigation made the condition a plain one, and it is justifiable to assume some direct causal relation between a double pneumonia with rusty sputum, that is found rich in diplococci, and bacterial deposits on the cardiac valves, both arising simultaneously.

I herewith close this short contribution on the blood-conditions in septic individuals, and, coming back to my original view, beg to state that the experiments that have been carried on in many lines give a clear picture not only of single elements in the blood, but also of the general effect of the changes produced upon the blood as a whole by septic processes. I believe, furthermore, that I am justified in stating that septic diseases artificially brought on in animals, as by the injection of decomposing masses or pyogenic bacteria, and the recorded results of these experiments cannot be indiscriminately applied to the diseases of man, but that we must attempt here, too, as in the study of other chapters in human pathology, to seek information, as far as this is possible, in observations made on the human subject.

In conclusion I will give a short *résumé* of the most important facts elicited above.

CONCLUSIONS.

1. For systematic examination of the blood it is desirable, in order to obtain the fluid in large quantities, and without the admixture of tissue-serum, to abstract it from a vein by puncture; and, in order to study its quality, not to confine the examination to the number of corpuscular elements existing, but to discover in addition the concentration of the blood itself and of its isolated serum.

2. In all cases of sepsis and allied conditions (endocarditis ulcerosa)

there is a considerable reduction in the concentration of the blood, a much greater reduction, on an average, than in other acute infectious diseases.

3. This dilution of the blood may take place very rapidly in severe cases, and may reach a considerable degree within a few hours after the onset of symptoms; the increase in dilution progresses proportionately to the duration of the disease and the severity of the symptoms.

4. The dilution of the blood is due to a loss of albumin from the blood proper, though especially from the serum, and is brought about apparently by an increased destruction of red corpuscles, but also by a transudation of fluid from the tissues into the blood, a result of the lymphagogue action of the metabolic products of pyogenic bacteria.

5. Every case in which a rapid reduction of blood-residue to fourteen per cent. existed, although life was not directly threatened, succumbed to a fatal issue in the course of the disease. In the most severe cases the residue of the blood sank to half the normal quantity, and even lower.

6. The number of red corpuscles was diminished in all cases; the hæmoglobin was diminished approximately in proportion to this reduction, and in the very severe cases only there were found the morphological changes of poikilocytosis, microcytosis, and macrocytosis.

7. The leucocytes were increased in every case, the polynuclear neutrophil forms being especially involved in this increase. No relation could be pointed out between the degree of leucocytosis and the severity of the disease.

8. The bacteriological examination was successful in establishing the existence of pyogenic bacteria in the blood of one case of puerperal sepsis and several cases of endocarditis ulcerosa; in the main, pyogenic staphylococci and streptococci were found, and in one case the diplococcus pneumoniae (Fränkel), as verified by a post-mortem examination of the ulcerated cardiac valves. For bacteriological examination, also, puncture of a vein and direct reception of the blood in tubes containing culture media is to be recommended.



FIG. 1.

A CASE OF AKROME GALY.

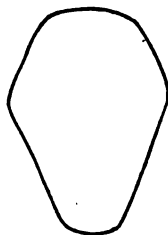
CLINICAL LECTURE DELIVERED AT THE POLYCLINIC HOSPITAL, PHILADELPHIA.

BY SOLOMON SOLIS-COHEN, M.D.,

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GENTLEMEN,—The diagnosis of the case before us may be made by inspection of the patient alone. The first thing that attracts attention is his attitude. You notice that his head is bent forward and downward, and that the position thus given to his neck is the continuation of a spinal curve extending to the upper portion of the dorsal region. (Fig. 1.) In other words, the patient has cervico-dorsal kyphosis ; and, if we now ask him to strip so as to expose the entire back and abdomen, you notice that he has also an exaggeration of the normal dorso-lumbar curve,—a slight lordosis, giving a peculiar “Punch”-like projection to the abdomen. Asking him to elevate the face, our attention is directed to several peculiarities of the features. (Fig. 2.) The nose is very large ; the lower jaw is long and broad, and, as you see when we expose the teeth, projects somewhat beyond the plane of the upper jaw. The lips are thick and the lower lip is overhanging. The superciliary ridges are markedly enlarged, standing out like great knobs from the forehead and giving the eyes a somewhat sunken appearance. The malar bones are likewise enlarged and projecting, so that the outline of the face assumes somewhat the shape that I trace upon the board (Fig. 3),—an irregular oval, having angles instead of curves. Continuing our inspection of the patient, we notice that the sternal ends of the clavicles are slightly enlarged, and that, owing partly to the peculiar position of his head, there appears to be a great hollow space above the sternum, between that bone and the cartilaginous framework of the larynx and trachea. But there are two other causes contributing

FIG. 3.



Outline of face in akromegaly.

to this peculiar appearance,—one is actual enlargement of the clavicles pushing the sternum forward and outward; the other, the absence of a structure that normally assists in preserving the rotundity of outline of the neck in this situation,—namely, the thyroid gland. You may remember what I have said to you upon several occasions concerning the difficulty that I at one time experienced in satisfying myself as to the presence or absence of this organ, its enlargement or diminution in size; and also that I now feel confident of my ability to determine these facts by palpation. You observe that I place my index and middle fingers, like the two prongs of a fork, straddling the larynx, at the level of the cricoid cartilage, or at the laryngo-tracheal junction, just below the cricoid cartilage, and tell the patient to swallow. This motion causes the larynx and trachea to rise, bearing with them the thyroid gland; and, as the structures glide between the sides of my fingers, I am able to appreciate, in normal subjects, the presence of the lateral lobes of the thyroid gland and to estimate their size and consistency. Placing the pulp of one finger below the cricoid cartilage, and telling the patient to swallow, the isthmus of the thyroid gland may be palpated. Since I have made it an invariable rule to examine every patient—no matter what the disease—in this manner, I have acquired a feeling of certainty as to the results of examination, which was previously wanting, on account of the infrequency with which the examination was made in subjects not presenting enlargement of the organ or enlargement of other structure liable to be mistaken for goitre. Now, in this subject I fail to get any indication of the isthmus of the thyroid, and, while I cannot affirm positively that the lateral lobes are wanting, I am able to say that, if present, they are not normal in size, as they give no positive indication of their presence.

But I have not finished recording the information to be obtained upon simple inspection of this patient. Notice his breathing. There is absolutely no expansion of the chest to be detected by the eye. Placing my hands upon opposite sides of the thorax, I feel no expansive movement. The thorax slightly rises and falls as a whole; the abdomen protrudes and recedes, indicating that breathing is entirely diaphragmatic. We attempt to verify this observation by measurement. We find that upon forced expiration the circumference of the chest at the level of the nipples is ninety centimetres, and in forced inspiration it is ninety-one centimetres. The circumference of the chest at the ensiform cartilage in forced expiration is eighty-seven and a half centimetres, and in forced inspiration eighty-eight and a half centimetres. In other

words, there is an expansion of but one centimetre, hardly one-sixth of the normal expansion; while even this slight expansion that we have noted is not produced except upon extraordinary effort. Investigating the cause of this want of motion of the chest, we find that it is not due to over-distention, as in emphysema, for the extreme measurement is not great, and you will notice that there is an appearance of lateral compression of the lower part of the chest, quite different from the barrel-shaped enlargement observed in cases of emphysema. Percussion, too, fails to elicit the hyper-resonance which would be found in a case of over-distention of the alveoli. The ribs are thickened, broadened; brought into close contact with one another. On attempting to mark out the intercostal spaces, we find, from the sternum to mid-axilla, great resistance offered to the palpating finger, indicating either that the ribs are in actual apposition or that a bridge of bony or fibrous tissue has been thrown out between rib and rib. The costal cartilages give a sensation of ossification, so that the anterior chest wall is, as it were, a complete bony cuirass, almost immovable. The skin and adherent subcutaneous tissue of the abdomen above the umbilicus are thrown into thick transverse ridges, between which lie deep furrows. The chest and arms present marked hairiness. The hands offer some interesting points for study; they are broad, somewhat spade-shaped; the fingers are thick and stumpy, yet with bony enlargements at the knuckles and finger-joints, which are also somewhat distorted. This appearance of the joints, the patient says, is not uncommon in men engaged, as he is, in boiler-making, and we cannot attach any importance to it. Some of the smaller phalangeal articulations, however, present an appearance not unlike that seen in rheumatoid arthritis, the change being greater than can altogether be accounted for by occupation. The furrows of the hand and of all the finger-joints are abnormally deepened. The skin is thick and coarse; the markings coarse-grained. The ends of the fingers form thick pads; the nails are broad, flat, and longitudinally ridged. The veins of the arms are prominent, tortuous, and rigid. The arteries, too, are somewhat thickened and rigid. We note a distinct pulsation of the arteries at the bend of the elbow.

As we now cast our gaze upward we see, likewise, pulsation of the arteries of the neck; and, examining more closely the skin of the face, we find it to be thick and heavy; we note that especially upon the forehead it lies in marked folds like those of the abdomen. It has a greasy appearance and feel. The ears are large, projecting, and the cartilages somewhat rigid. The teeth are bad.

Now let us look at his lower extremities. We note that the gluteal masses are much wasted; that on the right side shows a projecting lump, which appears to be a fatty tumor. It is possible, however, that this may be a myxomatous growth. All of the muscles are somewhat wasted, and the man tells us that he is losing strength. The veins of the legs, like those of the arms, are large, rigid, tortuous. The toes are distorted, the big toes of both feet being very large. There is a marked projection of the heel, somewhat like that of the negro, and there are decided fleshy pads on the outside of the feet, and especially beneath the big toe. Upon inquiry we learn that the patient has had to get his shoes of a broader size within the last three years. He has likewise had to get a larger hat within the same period, and has had to increase again within the last year. The hair of his head is thick and coarse, and we notice that the parting at the back is far towards the right.

Looking at his back more closely, we notice that the shoulder-blades are prominent, the spinous processes apparently enlarged, and that there is also, in addition to the curvatures already spoken of, a lateral curvature to the left of the middle line in the dorsal region, thrusting the left shoulder higher than the right.

The man is fifty-three years of age, is married, and gives a history of venereal disease. You see I know a little more about him than I have told you to-day, for he first appeared before us more than a year ago, on the 30th of November, 1892, and after that was under observation for some months. Being improved by treatment, he disappeared, and now has returned for relief of symptoms differing from those first presented. But I desired that you should make a diagnosis simply upon what you saw, and I have been pleased to hear a member of the class reply to my inquiry as to the nature of the case, that it is one of akromegaly.

This peculiar affection, first described by Marie from his studies of patients in the wards of Charcot, was at one time considered to be very rare; but the cases now accumulating in literature must cause this opinion to be materially modified. The truth is, I believe, as in so many other affections, that it has simply been unrecognized until the genius of an acute observer pointed it out and made it easy for all others to follow in the same line of study.

This is the second case that has come, in a certain sense accidentally, under my own personal notice. The first one, which I reported to the College of Physicians of Philadelphia, and of which I now exhibit to you photographs (Figs. 4 and 5), was that of a young man whom, whilst visiting a New England college, I happened to see engaged in



FIG. 2.—Akromegaly. Thos. F., aged 52 years.



FIG. 4.—Early akromegaly. X. Y. Z., aged 25 years.



FIG. 5.—Early akromegaly. X. Y. Z., aged 25 years.



FIG. 6.—Thos. F., aged 30. Changes of akromegaly not present.



FIG. 7.—Early changes in akromegaly. Thos. F., aged 47 years.

athletic exercises. That man had an enormous thoracic expansion—over sixteen centimetres.

The patient before us originally applied to this clinic for treatment for nothing obviously connected with the conditions which I have thus far described, and it was simply because our attention had been directed to the subject that we recognized that we had more to deal with than what the man complained of, and that his appearance indicated something else than simple ugliness. Before reading to you such portions of our earlier notes as bear upon matters not yet developed in to-day's examination, and in order that you may appreciate the extent of the changes which our patient's face and figure have undergone, I exhibit to you copies of photographs of himself taken at the ages of thirty and of forty-seven respectively (Figs. 6 and 7).

You observe that this is a progressive condition, and that its development in our patient began somewhat past the middle of life.

And now let us take up the case from the stand-point of the previous history of the patient and the development of the signs we have noted and their accompanying symptoms. The notes which I now read are dated November 30, 1892.

The patient complains of pain over the eyes, which darts through to the back of his head. This has continued for seven months and is steadily getting worse. The headache has for some weeks been almost continuous, and so severe at times that the patient cannot lie down to sleep, on account of the aggravation caused by pressure. At this examination, however, the scalp is but slightly tender. At times he loses his vision, vertigo occurs, and if he is walking he staggers. He is also annoyed by excessive urination, having to rise two or three times every night to make water; and, though he emptied his bladder about an hour before coming into the clinic, he now passes nearly a pint of water without difficulty. The urine is light-colored, 1010 specific gravity, acid in reaction, and on microscopic and routine chemical examination shows nothing abnormal.

The patient exhibits some degree of mental hebetude, part of which may be due to his deafness, the latter being probably the result of his occupation. He is a native of Ireland, and at the age of ten came to this country. At that time he appears to have been perfectly well physically and mentally, and began earning his living. About one year ago he began to feel weak and out of sorts; he was forgetful of things that he intended to do, and had to be reminded of them.

About the same time he began to complain of unusual drowsiness; he also states that he had an attack of pneumonia a year ago, but we

cannot learn whether this preceded or followed the other symptoms of which he complains.

His appetite is more than good ; he often feels hungry, and rarely rises from the table satisfied. He states that he does not drink an excessive amount of water and is not troubled with thirst.

As regards his disposition, he appears to be very good-natured, if a little stupid (you will remember that I have told you that he is deaf, and consequently does not hear what I am saying) ; and he states on inquiry that he is not and has never been irritable.

His voice is deep, having peculiar intonation, only partially due to his deafness. His speech is slow, with thick articulation, and a tendency to chop off the last syllables. His tongue is thick, deeply furrowed, and somewhat coated. His bowels are usually constipated, and he has slight dyspeptic symptoms.

Concerning the change in his form and features, he states that he first noticed about three years ago that his back was curved, and that the change in the features became sufficiently marked to attract attention at about the same time.

As to previous ailments of the patient, we learn that when nine months of age he fell upon his head and was sick for some time. He can, of course, give no definite account of this, but remembers that when he was older he was unable to bear noise, and for that reason he was not allowed to go to school, so that now he can neither read nor write. This defective education has rendered somewhat more difficult the examination of the eyes, kindly undertaken by my colleague, Professor Jackson, who, however, finds no significant error of vision, or of color-sense, or lesion of the fundus.

We learn that the patient had measles and varioloid when a child, and chancre so many years ago that he cannot recollect the exact date. We can get no history of later symptoms of syphilis.

His father and mother died of old age. Two sisters died of phthisis and one of tumor of unknown nature, probably cancer. He has one brother and two sisters living and well.

There is no family history of nervous trouble. He has a daughter and a grand-daughter, both of whom he says are well, giving no evidence of chronic disease of any nature. The daughter has a very long and broad chin, which markedly projects.

The following is the record of auscultation and percussion of the lungs and heart made at the time of the patient's first visit :

The apex of the lung does not extend above the clavicle on either side. The resonance is impaired upon both sides anteriorly and in the

axilla, more upon the right than upon the left. Towards the base on both sides the resonance becomes a little clearer; and on the left side, as we get below the area of cardiac dulness, stomach tympany and lung resonance are intermingled as far as the border of the ribs. Posteriorly, at the upper portion of both sides, there is impaired resonance; but good pulmonary resonance begins about the middle of the back and extends lower than is usual, seeming to go below the bony framework. Anteriorly the breathing is quite bronchial at the apices, but less so towards the lower portion of the chest. Posteriorly the breath-sounds are rough above the inferior angles of the scapulæ and puerile below that.

The apex beat of the heart is at the sixth interspace and nipple line, the visible pulsation extending a little beyond the latter boundary.

A slight thrill and pulsation are perceptible on palpation at the seventh interspace. The cardiac dulness begins at the fourth interspace, extending to the nipple line from the right edge of the sternum. At mid-sternum it ceases between the fifth and sixth costo-sternal articulations and gradually contracts to the apex.

The first sound of the heart is feeble and impure; the second sound accentuated. There is no murmur at the aortic or pulmonary cartilage.

The following are the measurements of the various portions of the patient's body, taken to-day just before his appearance at the clinic, February 3, 1894:

HEAD, FACE, ETC.

	Centimetres.
Circumference—"occipito-frontal"	58
Circumference—"mento-bregmatic"	61
Circumference—"trachelo-bregmatic"	58
Circumference of mandible (from angle to angle around front) . .	24
Circumference from temporal root of zygoma around point of chin to temporal root on opposite side	84
From zygoma (temporal root) over root of nose to same point on opposite side	26
Depth of jaw (from upper margin of lower lip)	5
Circumference over tip from ala to ala	8
Root of nose to hair line on forehead	8
Circumference from "temporal fossa to temporal fossa" across forehead, above superciliary ridge	20
Length of nose (root to tip)	6½
Depth of ramus of jaw	9
Length of left ear	7½
Length of right ear	7½
Circumference of neck	38½
Bottom of supra-sternal notch to tip of ensiform	24½
Distance between acromions measured over back	84

	Centimetres.
Circumference of chest at nipples (forced expiration)	90
Circumference of chest at nipples (forced inspiration)	91
Circumference of chest at ensiform (forced expiration)	87½
Circumference of chest at ensiform (forced inspiration)	88½
Circumference of abdomen (most dependent portion nine centimetres below umbilicus and from above crests of ilia)	92

Patient has kyphosis and lordosis (exaggeration of normal dorsal and dorso-lumbar curves).

Spinal column also curves to left of mid-line in dorsal region.

	Centimetres.
Height of this curve	2
Length of this curve	18
Length of spine (from occiput to sacrum)	65½
Point at which curve begins, seventh cervical vertebra.	
Deviation of spine by plumb-line	6

The patient's height is 1.76+ metres (nearly five feet nine and one-half inches). Before he became crooked it was about 1.78 metres (five feet ten inches). As the projection of the curve is greater than the diminution in height, there is evident lengthening of the spinal column as a whole, either from enlargement of vertebræ or thickening of inter-vertebral cartilages, or both.

HAND AND ARM.

	Centimetres.	Centimetres.
	Right.	Left.
From radio-carpal articulation to end of middle finger	21½	21½
From radio-carpal articulation to proximal end of middle finger	10	10
Circumference of hand at heads of metacarpal bones	24½	24½
Circumference of wrist	19½	19
Circumference of arm at centre of biceps	26½	25½
Circumference of forearm (junction of upper and middle third)	27½	26½
From tip of acromion to external condyle of humerus		38½
From olecranon to styloid process of radius		29½
Breadth of hand, including head of metacarpal bone of thumb		18
Circumference of metacarpus, including that of thumb	27	26½
Length of thumb	7½	...
Length of middle finger	12	...
Circumference of last phalangeal joint of middle finger	8½	...
Circumference of joint between first and second phalanges of middle finger	8	...
Circumference of forefinger (proximal phalanx)	8	8

FOOT AND LEG.

	Centimetres.	
	Right.	Left.
Length of foot, heel to tip of great toe	26½	...
Circumference of foot at ball of great toe	28	27½
Circumference of foot at instep (scaphoid bone)	26½	26
Circumference of foot across instep and heel	40½	40
Circumference of ankle (narrowest part)	22	22
Circumference of leg at junction of upper and middle third of largest part of calf	32	33
Circumference of thigh at junction of upper and middle third	46½	46½
Circumference of right knee (leg extended, across patella)	37½	37½
From anterior superior spine to interior condyle of femur	51½	52
Circumference of phalangeal joint of big toe	11	...
Length of big toe	8½	8
From centre of interior malleolus around point of heel to centre of external	24	25
From head of fibula to bottom of external malleolus	40½	40

The muscles generally appear to be wasted and exhibit decreased quantitative reaction to galvanism, but no polar change. The knee-jerks are lessened. Station with the eyes closed is good. Tactile sensation is generally impaired, but sensation as to heat and cold unimpaired. Our notes state that while the patient was under observation he passed about five pints of urine daily; some days nearly eight pints. No albumin, no tube-cast, no sugar, no excess of urates or uric acid, was ever found in it. Sexual power is preserved.

His headache and the paroxysmal flushing and sweating that he complained of were markedly relieved under treatment with *picrotoxin*, one-sixtieth of a grain, three times a day. Later they returned, but quickly disappeared under treatment with desiccated thyroid gland, fifteen grains daily in a single dose having been given for three weeks. This is a somewhat larger dose than I should now be inclined to use. At his last visit to the clinic he was given a quantity of the powder, which he continued to take in an irregular manner for a month or six weeks. He has been without medicine for nearly a year.

He comes back now complaining of intense pain along the course of the great sciatic nerve of the right side. This has been of gradual development, and during the last few weeks has been unbearable. The track of the nerve is sensitive to pressure. There is evidently a neuritis. His headaches have not returned. His sexual power is still preserved. His memory is worse; drowsiness is still marked; he falls asleep very easily. He has been working whenever he could get work

to do. He says that he feels much more supple than he did when he first came to the clinic, and that he can now wear a pair of gloves (part of the uniform of some secret order) that were bought some years ago and afterwards were too small.

We shall again resort to treatment with desiccated thyroid gland, giving him five grains in capsule night and morning, and see whether or not it will affect the present symptoms.¹ We shall have him measure his urine and will examine it carefully, and will also try to observe carefully what effect the thyroid powder has upon it. I have noted in other cases that thyroid extract has a decided diuretic power. This man has already polyuria, so that anything which would markedly increase diuresis would be extremely disagreeable to him. But it is just possible that as the picrotoxin diminished his excessive urination in our previous observation of him, the thyroid extract will have a similar effect² now, for I believe the opinion to be justified that many of the symptoms of akromegaly are due to deficiency of thyroid secretion, as is the case in myxœdema; and just as in myxœdema the symptoms are relieved by an artificial thyroid secretion, so in a case of akromegaly we can likewise expect improvement from the same measure.

Before dismissing the subject let me say a few words about the general character of this curious affection, and point out a few particulars in which the case before us differs from others.

Akromegaly evidently belongs to that great and little-understood group of tropho-neuroses associated with abnormality of the thyroid gland. It is characterized by abnormal exaggeration of the growth of bony tissue, chiefly in the face and the extremities, but as my two cases, with others, prove, in the chest and elsewhere as well. There is likewise overgrowth of the related soft parts, eventually accompanied, however, by loss of muscular power indicating a state of malnutrition that may even progress to atrophy. In our case there is evident atrophy of the arm and thigh muscles. The heart, likewise, appears to be enlarged and weakened. Thus there is a diversion of nutrition to bone and subcutaneous tissues at the expense of muscle. Associated with this are certain vascular abnormalities, especially varicosities of veins, due, of course, to impaired nutrition. In the case before us there are joint changes. This is unusual; although a connection between thyroid disease and rheumatoid arthritis has apparently been established.

¹ The sciatic pain disappeared entirely in the course of two weeks.

² This was the case, the quantity of urine falling from five quarts to three quarts, the specific gravity being increased to 1015-18. June 15, 1894, the report is: quantity two quarts, reaction acid, specific gravity 1024, no albumin, no sugar.

The skin apparently shows hypertrophy; yet this is rather of its less distinctive structures. It is an overgrowth, but not a heightened nutrition.

There is some impairment of mental power, showing a further resemblance with myxedema and cretinism. The vaso-motor disturbances likewise bring into consideration a relationship with exophthalmic goitre and those other varieties of vaso-motor ataxia of which we have seen at this clinic so many examples.

In the case before us there is not only abnormal flushing, vertigo, and transient blindness, but also polyuria. This is not due to interstitial nephritis, for we have no other symptom of that condition, and the urine does not indicate it. Polyuria is frequent in cases of akromegaly, and in some instances glycosuria has been observed. Here the disease touches another great group of metabolic disorders, of which diabetes mellitus is the most prominent type. Diabetes mellitus, we know, sometimes depends upon the uric-acid diathesis, and lithæmia is one of the chief factors in exciting certain vaso-motor disorders; on the other hand, we know that certain cases of diabetes are dependent upon disease of the pancreas, with which disease of the solar plexus is often associated. All of these facts together seem to point, first, to the sympathetic or organic nervous system, the nervous system of vegetative life or nutrition, as the remote seat of the disorder. (The absence of gross lesion at autopsy does not necessarily prove these structures to have functionated properly during life.) Secondly, they indicate that the thyroid gland has a complex function to perform in regulating nutrition, and that the trophic changes noted are in large measure dependent upon failure of that function. It may be secretory, it may be depurative, or it may be both; the facts yet before us do not warrant a positive opinion. It is highly probable that more exact chemical, or what I may term toxico-biological, studies of the blood, the urine, and the sweat in cases of akromegaly and allied disorders will reveal the presence of one or more toxins to which many of the symptoms may be attributed. This, however, will not explain the origin of the disorder, which must be sought in the visceral or organic nervous system. It is most probably congenital in origin but of slow development. Or perhaps it would be better to say that it depends upon failures of development or early retrogressive changes, the effects of which are manifested only at a certain period of individual evolution.

Still more strongly suggestive of the fundamental relation of thyroid failure with the trophic changes of akromegaly are certain symptoms not presented by our patient, but found in many cases. I refer to

persistent headache and to hemianopsia and other forms of perverted or imperfect vision. You fail to see at once what the connection is. It is this: in cases presenting these symptoms there has been found, *post mortem*, an enlargement of the pituitary body. At first this lesion was thought to be constant and to have a causative connection with the disorder generally; but now we know that it is not constant. The situation of the pituitary body causes it in its overgrowth to press upon neighboring cerebral structures, upon the optic nerves and optic commissure, and thus mechanically to cause the visual symptoms and headache as epiphenomena. The portion of the pituitary that enlarges is the anterior part, that developed from the alimentary tract, and embryologically related with the thyroid gland. It may be that this enlargement is in the nature of a compensatory hypertrophy doing harm only because of the situation of the pituitary body; or it may be that the central organic nervous defect causes both the thyroid and the pituitary changes. I am more inclined to the former view because of the effect of thyroid medication in relieving our patient's headache, possibly by checking pituitary overgrowth. The absence of hemianopsia indicates that there has never been marked lesion of that body.

In conclusion I will briefly summarize for you the characteristic signs and symptoms of akromegaly upon which a diagnosis can be based:

1. *Enlargement of the bones, fibro-cartilages, and overlying soft parts, especially of the face, hands, and feet.* The face is elliptical in outline, the lower jaw projects; the hands are broadened more than they are lengthened, are spade-shaped, with sausage-like fingers; the heels project and fleshy pads are found on the outer aspect of the feet. 2. *Spinal curvature*, causing hanging of the head with protuberance of the abdomen, and sometimes related with marked increase of stature. 3. *Enlargement or atrophy of the thyroid gland.*

In addition to these cardinal symptoms there is frequently enlargement and hardening of the cartilages of the larynx, trachea, and ribs, and pigmentation of the skin. Thirst, boulimia, and polyuria are common. Many cases present hemianopsia, irregular limitation of visual field, or blindness. Deafness is not rare. Drowsiness and headache are often marked.

IDIOPATHIC ENLARGEMENT OF THE HEART.

CLINICAL LECTURE DELIVERED AT THE POST-GRADUATE MEDICAL SCHOOL.

BY ROBERT H. BABCOCK, M.D., CHICAGO,

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GENTLEMEN,—The patient is forty-nine years of age, a Swede by birth, and a machinist by occupation. His family history is good. He gives a history of the ordinary diseases of childhood, and considered himself in good health until two years ago. His maximum weight ten years ago was one hundred and seventy pounds, and, as his height is five feet six inches, he was, of course, of heavy build. He was always a hearty feeder, smoked inordinately, and drank moderately, taking his beer at noon, and Saturday nights and Sundays indulging in whiskey. He considered himself in perfect health with the exception that for two years prior to the date from which the present illness begins he had fallen off in appetite and ate but little. Two years ago, in March, while working at his trade in the shop, a belt became loose; he took a very heavy ladder and at great muscular effort placed it in position to adjust the belt. He was seized at once with shortness of breath, cough, and expectoration of considerable frothy mucus. He was obliged soon afterwards to go home, and was ill in bed for a week with what appears to have been œdema of the lower extremities, cough, and palpitation of the heart, and after a week's illness he again sought his work, but was obliged, because of the return of the symptoms, to again quit work. He has not worked from that time to this. His present symptoms are shortness of breath and great weakness. He cannot walk even two blocks because of dyspnoea, and to carry a scuttle of coal up-stairs completely exhausts him. He has no cough, no expectoration, and complains of no indigestion; nevertheless, upon rising in the morning, he vomits a considerable amount of mucus. His bowels are

regular. He has no œdema. Physical examination reveals some very interesting conditions.

The chest, as you see, is a broad, deep, round chest. The left nipple measures three and three-quarters inches from the left border of the sternum. . An examination of the lungs has shown them to be healthy ; percussion note resonant ; respiratory sounds vesicular ; no râles. Examination of the heart shows the apex beat to be in the fifth intercostal space, just outside the nipple line. It is feeble, being a gentle tap, but of irregular force. Within this oval area about the nipple the heart's impulse is irregular, heaving, and feeble. Percussion reveals great enlargement of the heart ; upon the left parasternal line dulness begins at the lower border of the second costal cartilage and passes downward until it blends with the liver in the fifth intercostal space ; at the right border, upon a level with the fourth costal cartilage, dulness begins one and three-quarters inches to the right of the sternum and passes across a total transverse distance of eight inches to at least an inch outside of the left mammary line. This gives a quadrangular area of abnormal extent. Upon auscultation the first sound is short, feeble, and irregular in force and intensity. The second sound is feeble, but the second aortic sound is accented relatively as compared with the second pulmonary sound. There are no murmurs. The first sound is muffled, but there is no distinct murmur. Liver dulness begins on the right mammary line at the lower border of the fifth rib and passes down fully to the lower edge of the costal arch, where the lower border of the liver can be felt firm, but it does not extend below the rib. The spleen is not palpable, and the area of splenic dulness is normal. The abdomen is full and not unduly tympanitic, and reveals nothing special. An analysis of the urine shows a total quantity of one thousand cubic centimetres in twenty-four hours ; specific gravity of 1006, of reddish-yellow color. There is a plain trace of albumin. Urea is greatly diminished during the twenty-four hours in which this specimen was collected. The total amount of urea was only three grammes, or forty-five grains. A microscopical examination of the urinary sediment reveals small hyaline and granular casts, few in number. Palpation of the pulse shows a small, rapid, arrhythmic pulse, running from 125 to 130 a minute, tense, but exceedingly irregular as to force and volume, there being occasionally strong beats which reach the finger, followed by a greater or less number of small beats quickly succeeding each other. We have then, in this case, two conditions of great interest,— we have renal cirrhosis, probably in the stage of contraction, and we have a very greatly enlarged heart, what is commonly called a dilated

heart, or what Fraentzel describes as idiopathic enlargement of the heart. The renal condition, although of great interest, is not what we propose to discuss this afternoon. It is the condition of the heart.

The sequence of conditions in this man's history is probably as follows: There was unquestionably cirrhosis of the kidneys with prolonged high arterial tension in consequence, and secondary hypertrophy of the left ventricle; then a history of strain, which caused the hypertrophied heart to become dilated. The dilatation of the heart which ensued aggravated the kidney lesion, and the chronic interstitial nephritis which exists has in turn aggravated the condition of the heart and prevented the re-establishment of compensation. We have here a vicious circle, namely, chronic interstitial nephritis with idiopathic enlargement of the heart, each increasing and aggravating the other.

Now, idiopathic enlargement of the heart, as Fraentzel designates it, affecting primarily the left ventricle, is due primarily to prolonged high arterial tension, and this increase of arterial tension may be secondary to (1) cirrhosis of the kidneys, (2) chronic arterio-sclerosis, (3) congenital narrowing of the arterial system, and (4) some obscure condition probably dependent upon defective assimilation and elimination, which we do not fully understand, connected probably with the circulation in the blood of toxins. I am speaking, of course, of idiopathic enlargement of the heart without valvular lesions. Prolonged high arterial tension is the chief factor concerned in the production of this form of enlargement of the heart. The enlargement may involve either the right side or the left side of the heart, or both. In this instance the enlargement was primarily of the left ventricle, and the enlargement which now exists of the right side of the heart is probably secondary to the dilatation which has taken place of the left ventricle. Prolonged high arterial tension, when independent of organic disease of the kidneys or blood-vessels, seems to be due to what the Germans call *luxus consumption*. We observe it in individuals who belong to the better class, who are more or less sedentary in occupation, who are hearty feeders, and oftentimes great diners-out. We see it in individuals who have ample chest and ample or highly protuberant abdomens. High arterial tension is also produced by uric acid circulating in the blood, or uric-acidæmia. Then we see this condition of high arterial tension in the working classes, and Fraentzel seems to think it is due to the strenuous and continuous labor to which these individuals are subjected. He thinks it is also produced by the excessive use of tobacco, as excessive smoking, and also by the abuse of alcoholic stimulants. The first effect of prolonged high arterial tension is to cause hyper-

trophy of the wall of the left ventricle. The increased blood-pressure acts as a barrier to the free circulation within the arterial system; the left ventricle, therefore, takes on a degree of compensatory hypertrophy sufficient to enable it to carry on the circulation. So long as hypertrophy of the ventricular wall exists it is compensatory, but there comes a time in most of these individuals, sooner or later, when, due to lessened nutrition, the heart under increased strain, perhaps sudden strain, or it may be prolonged strain, yields to the increased internal pressure and dilates.

In this man we have a history which leads us to infer the existence of prolonged high arterial tension, probably secondary to chronic interstitial nephritis. For two years prior to the beginning of his present symptoms, his nutrition was failing, since, as he states, his appetite had fallen off and he took but little nourishment. The heart-muscle began to suffer in its nutrition, and one day, under a violent strain, the internal blood-pressure was too much for the resistance of the ventricular wall, and dilatation supervened. There was acute dilatation of the left ventricle. Symptoms began at once, as dyspnoea and cough. Probably, in consequence of unfavorable hygienic conditions, as well as the persistent, prolonged, high arterial tension, resulting from the renal diseases, the heart has been unable to regain its former compensation, and the symptoms are therefore due to the persistence of dilatation. His symptoms at present are not marked, but there will come a time in all such cases when urgent symptoms are produced, when the dyspnoea becomes so extreme as to amount to actual orthopnoea, and the increased tension within the venous system (the engorgement of the venous system) leads to pronounced disturbance of the functions of all the organs and to renal congestion, and sooner or later to the establishment of oedema. The symptoms are those of passive congestion, cough, with sero-mucous—sometimes bloody—expectoration, great cardiac dyspnoea, more or less insomnia, or perhaps somnolence, great disturbance of digestion, leading to fermentation within the digestive tract, to consequent distention of the stomach and pressure upward against the already dilated right ventricle, engorgement of the portal system, leading perhaps to ascites, obstruction to the venous and lymphatic circulation, leading to oedema of the lower extremities; this picture of distress growing worse and worse until finally the heart stops from either exhaustion or paralysis, or possibly the patient's life is terminated by pulmonary oedema.

The morbid anatomy of these cases is extremely interesting, and is beautifully illustrated by an autopsy which I had the other evening in the

case of a patient who died of dilatation of the heart in consequence of prolonged high arterial tension ; in other words, an idiopathic enlargement of the heart. The heart was found enormously enlarged. Although in a large individual, a man who weighed two hundred and eighteen pounds, still the heart was perhaps twice the size of his fist ; it must have weighed sixteen or eighteen ounces. The pericardium showed no evidences of previous inflammation, and contained a little transuded serum. The right ventricle was thin and greatly dilated, containing post-mortem clots. The muscle was pale and anæmic, but did not give macroscopic evidences of fatty degeneration. The left ventricle was also enormously dilated, its wall three-quarters of an inch in thickness, with its chamber filled with post-mortem clots. There were also no macroscopic appearances of fatty degeneration. All of the valves were healthy, and the aortic and pulmonary valves stood the water test. The aorta showed no trace of chronic arterio-sclerosis. There was one small patch of sclerosis in the left coronary artery about half an inch from its mouth, but it was not sufficient to occlude the vessel in the least. Both pleural cavities contained serum, perhaps a quart in all, and the lower lobe of the right lung was in a state of hypostatic congestion. The liver was greatly engorged, but not fatty. The spleen was also congested. The kidneys were in a state of chronic engorgement, not cirrhosis, although the left kidney was considerably larger than the right and contained numerous small cysts, together with streaks of what looked like fibroid tissue. Sections will be made of both these organs, and the heart.

That case beautifully illustrated the morbid anatomy of idiopathic enlargement of the heart. In many instances there is a pronounced or variable degree of chronic arterio-sclerosis associated with perhaps cirrhosis of the kidney, but the case I refer to illustrated very beautifully those examples of idiopathic enlargement of the heart due to prolonged high arterial tension without organic disease of blood-vessels or kidneys to explain the cardiac enlargement. That individual was a man, fifty-five years of age, who weighed two hundred and eighteen pounds, and who had been in perfect health up to about two months ago, since which time he had developed attacks of cardiac asthma, coming on at night or whenever he fell asleep. There was to be obtained by careful questioning a history of strain upon the heart two years ago in consequence of his having run a foot-race in a friendly contest. He was a hearty feeder, of sedentary pursuits, exactly the type of individual where prolonged high arterial tension is the result of *luxus consumption*. The symptoms are those of loss of equilibrium between the venous and the

arterial circulations. The arterial system becomes relatively depleted and the venous system relatively engorged. The pulse is commonly arrhythmic, irregular in force and volume as well as accelerated, yet the individual whose autopsy I just narrated had a pulse which was small and empty, was not particularly accelerated, rarely beating above ninety in the minute, and it was not arrhythmic. The heart in that instance evidently stopped in diastole; in other words, the patient died from paralysis of the left ventricle.

The diagnosis of these cases is not ordinarily difficult, yet if one were to depend entirely upon percussion he might in many cases be deceived. The area of cardiac dulness in many instances does not pass beyond the extreme limits which are assigned to the normal heart; that is, cardiac dulness in many cases does not pass to the left of the mammary line, yet the individuals commonly have such ample chests that the distance from the sternum to the nipple line is sufficient of itself to indicate an enlarged heart. In this present patient the left nipple is three and three-quarters inches from the left border of the sternum, and yet the cardiac dulness passes an inch farther to the left. The total transverse area is increased, passing also too far to the right. Diagnosis, therefore, depends upon the evidences by careful percussion of enlargement of the heart; while upon auscultation there is discovered no evidence of valvular lesion. In this individual the heart-sounds are faint and muffled, the first sound having lost its muscular or booming element, and the second aortic sound revealing high arterial tension through its accentuation. Then from the history of the habits and occupation of the patient, which of themselves would induce prolonged high arterial tension, or by the discovery of some condition within the arteries or the kidneys, as chronic arterio-sclerosis or chronic interstitial nephritis, which would lead to prolonged high arterial tension, and by the symptoms of respiratory and circulatory embarrassment with the rapid, feeble, arrhythmic pulse, we are able to determine that the case is one of cardiac enfeeblement, due to dilatation of what has previously been an hypertrophied heart.



To show the character of the pulse I will pass around some sphygmographic tracings, which beautifully illustrate the irregularity of the pulse, its smallness, and also its tension.

The prognosis in these cases is variable. Under proper treatment, instituted in time in the early history of the disease, it is possible to re-establish a tolerable degree of compensation; in fact, many patients are able to return to work. In other cases the ability to work is permanently impaired, and if the patients are to enjoy any degree of comfort it is at the expense of great carefulness and abstinence from much physical exercise and from labor. Then the prognosis is influenced also largely by the etiological factor concerned,—if the prolonged high arterial tension is in consequence of *luxus consumption*, or of changes within the kidneys or blood-vessels. What is the treatment of these cases?

The treatment may be divided into, first, the treatment of the stage of loss of compensation, the stage in which the heart is at first broken down in its resistance. Rest is the important factor along with cardiac stimulants and the administration of remedies calculated to decrease the high arterial tension. These patients usually present themselves with dyspnoea, cough and frothy expectoration, and an extremely rapid and feeble pulse, with all the evidences of cardiac dilatation. Our first indication, therefore, is to relieve the over-distended left ventricle and to whip the heart on to increased vigor or contraction. Hydragogue cathartics are, therefore, indicated from the first, since their action is to lessen arterial tension, and by preference I administer one of the mercurials, since the effect of calomel or blue mass is to lessen arterial tension even before its effects are manifested on the intestines, and this is followed by a saline. Digitalis, strophanthus, and their congeners are not suitable to these cases in the stage of loss of compensation, since the effect of digitalis, and to a less degree strophanthus, is to increase the resistance within the arterial system, and thereby increase the strain, already too much for the impaired right ventricle. It is preferable, therefore, to administer an arterial stimulant rather than one of the so-called cardiac tonics. Fraentzel's favorite combination is tincture of castor and ammoniated tincture of valerian. The diffusible stimulants, like camphor, musk, ether, and ammonia, and alcoholic stimulants are also in order. Under the effect of rest and cardiac stimulants improvement in the condition is generally manifested speedily, and if the diet be carefully regulated, the patient being at first limited to a milk diet, the urgency of the symptoms will commonly pass by in the next few days. As the patients regain strength and are able to be about, they should be cautioned against much exercise; in fact, the dyspnoea they experience prevents their taking too active exercise at first. However, it is better for them to walk quietly on a level surface (level ground)

rather than to remain too persistently in bed after the urgency of the symptoms has passed. As the patients regain their ability for exercise attempts may be made to strengthen the heart-muscle by the prolonged administration of strychnine salts, and also by the judicious use of gymnastics. Swedish movements properly given have become a popular form of exercise for the heart-muscle in Europe. Oertel's mountain climbing is not appropriate to these cases after compensation has been lost. Careful experimentation and testing of Oertel's method has demonstrated without doubt that it is highly beneficial for cases of threatened loss of compensation, cases in which hypertrophy still predominates over dilatation. When dilatation has once gained the ascendancy, great harm, even fatal consequences, may follow injudicious mountain climbing.

One of the most efficient means of treating these cases of idiopathic enlargement of the heart, even after compensation has been lost, is by what is known as the Schott Method, or the Balneological Gymnastic Method, a treatment by baths and gymnastic exercises. It is a treatment which has been in use in Germany for more than ten years, and which was elaborated by two German physicians by the name of Schott, and which is now carried on in its perfection at the watering place, Bad-Nauheim. It consists of saline baths of cool temperature, tepid, and baths which are charged with carbonic acid. They are weak at first, and the temperature is perhaps about 92° F., and the duration of the bath is from five to eight minutes. As the patients improve and the strength of the heart increases the baths are gradually strengthened in their saline constituents, and finally baths containing carbonic acid together with the salts are employed; at the same time the temperature is gradually lowered until patients are subjected to baths at a temperature of from 87° to 86° F. The duration is also lengthened until patients can endure baths of twenty minutes' duration. These baths are given daily with occasional intermissions. After three baths patients are given a day of rest, then three, and another day of rest, and so on. After each bath the patient is told to lie down for at least an hour. The effect is very marked. Subjectively the patient experiences a sensation of chilliness on entering the bath, and this chilly sensation usually passes away within a minute. The slight sense of dyspnoea gradually grows less, and although it may not disappear with the first or second bath, it does so after a few baths. The patient remains perfectly quiet immersed to his neck. After the first sensation of chilliness he experiences a delightful feeling of warmth or glow of the surface of the body. Any movement is followed by a sensation of chilli-

ness in the member which has been moved. Upon the heart and pulse the effect is very striking. The pulse becomes slow, stronger, and fuller. During my observation of the effects of these baths the past summer upon myself the pulse fell from eight to twenty beats in the minute, an average of perhaps fifteen beats in the minute. Previously it was small, weak, and irregular, but it became subsequently fuller, stronger, and more regular. Indeed, so strong and full did it become that before a bath of twenty minutes had terminated, the pulse was almost incompressible by the finger. The effect on the heart is to diminish the area of cardiac dulness. Careful percussion of the heart before and after the bath demonstrates actual diminution in the size of the cardiac area. It would seem as if the heart, beating more slowly and energetically, was the better able to empty the over-distended cavities and thereby to actually diminish in size. After the bath the effect persists for a considerable time, and if the patient lies down at once and rests, does not subject the heart to any undue effort, the pulse will remain slow, strong, and full, for from one to two or three hours. Cardiac murmurs, which were weak, almost inaudible before, become distinct after the bath, and conversely some murmurs which existed before will disappear after the bath when those murmurs have been due to cardiac dilatation, to a relative insufficiency, for instance, of the mitral valves. The heart sounds become stronger, and the undue accentuation of the second pulmonary sound diminishes, the increased strength of the second aortic sound indicating a better filling of the arterial system. In connection with these baths are given certain gymnastic exercises which consist of movements of extension, flexion, and rotation of the extremities and trunk, the principle of these exercises being that they must be exerted against resistance applied by an assistant. The exercises are made slowly and steadily without jerkiness, and with only such a degree of resistance as can be borne without producing undue acceleration of the pulse or respiratory embarrassment. The effect of these gymnastics is similar in kind, but slightly less in degree, to those produced by the baths. The pulse becomes slower, stronger, and fuller, and the over-distended heart will actually diminish in size after the judicious use of these exercises, as can be demonstrated by careful percussion.

Such a course of combined baths and gymnastics is given for an interval of perhaps seven or eight weeks. Then the patients are sent away to some salubrious resort, perhaps in the mountains, for a month, after which in very bad cases a second course of treatment is taken.

This Schott method of treatment is suitable to all forms of chronic

cardiac disease, excepting those in which heightened arterial tension would be disastrous, as in widespread and extreme chronic arteriosclerosis, aneurism of the aorta or other large vessels, or cardiac aneurism. Conditions in which valvular lesion exists, or in which the muscle of the heart has lost its tonicity,—that is, therefore, idiopathic enlargement of the heart,—are greatly benefited, and many times can be reinstated in perfect compensation; in fact, all conditions of the heart in which the muscle-fibre has retained sufficient integrity to be capable of regeneration are beneficially treated by this combination of baths and exercises.

I have had considerable experience this winter with the use of this system of cardiac therapeutics, since I have established some bath-rooms where I am treating a number of cases with very gratifying results. One case of idiopathic enlargement of the heart has experienced such a degree of improvement as to surprise not only the patient but myself. The patient is a male, about fifty years of age, whose heart was greatly dilated, particularly the left ventricle, the cardiac dulness extending considerably outside the left nipple, and the apex beat being merely a gentle and irregular tap. The pulse was exceedingly irregular, intermittent, and soft. After eight weeks of baths the patient expressed himself as free from all symptoms, and said he would not know he had a heart. To my great surprise the right side of the heart was normal in extent, and the left ventricle had contracted so that the dulness reached but little outside of the nipple line. The apex beat was on the nipple line and had become forcible and uniform in strength. The systolic murmur, which had existed at the apex, and been due to relative mitral incompetency, had almost disappeared; the first sound was fairly good in quality, and the second aortic sound had regained its relative strength.

In the case of our patient to-day these baths, unfortunately, are not indicated and would not promise much in the way of reinstating this damaged heart, because the chronic interstitial nephritis maintains the prolonged high arterial tension, and would serve, I fear, to constantly distend and dilate the weakened left ventricle. The patient has been treated here after the most approved fashion for the past two years by means of the usual remedies, and although his condition is to-day by no means what could be desired, the heart seems nevertheless to have gained somewhat in strength over its condition two years ago. He is now receiving, mainly, strychnine, together with some other cardiac tonics, I think small doses of strophanthus.

Neurology.

TWO LECTURES ON SOME FORMS OF MYELITIS AND OF SERIOUS SPINAL TRAUMATISM.

CLINICAL LECTURES DELIVERED AT THE PHILADELPHIA HOSPITAL.

BY CHARLES K. MILLS, M.D.,

Professor of Mental Diseases and of Medical Jurisprudence in the University of Pennsylvania; Neurologist to the Philadelphia Hospital.

LECTURE II.

Fracture—Dislocation and Compression Myelitis—Fracture and Hemorrhage into the Conus and Cauda Equina, and into the Lumbo-sacral Spinal Cord.

FRACTURE—DISLOCATION AND COMPRESSION MYELITIS IN THE DORSO-LUMBAR REGION.

CASE V.—This patient, I. S., aged forty-five, born in Ireland, in August, 1884, fell from a second-story window to the pavement, a distance of fourteen feet, striking her back at about the ninth or tenth dorsal vertebra. The spine was fractured. When she regained consciousness about seven hours after the accident, she had violent pains in the back and around the waist and was unable to move her legs. Sensation was lost below the seat of injury. She had also lost control of the bladder and rectum, and had to be catheterized during the subsequent four years. After that the urine dribbled away, constantly keeping her wet. She has even yet attacks of pain in her legs, which she describes as being in the bones, and also as sharp and shooting in character. Bed-sores have developed on the slightest provocation, and are very difficult to control; she has had a very large one in the ischio-sacral region.

She is not able to move either leg, neither has she foot or toe movements; and it is well to be specific in these statements, as she may have one and not the other. Both limbs are badly contracted at the knees, one nearly at a right angle, and the other at an angle of one hundred and twenty degrees. The limbs and feet are somewhat puffy, but this is not due to anasarca from kidney-trouble, but to deficient vaso-motor tonus. Upon her heel she has a sore,—a bed-sore, if you choose,—partly trophic and partly due to pressure. The toe-nails are ridged,

dark, and unhealthy looking, showing extremely bad nutrition. Her limbs are of good size, but I think this is due mostly to fat and to puffiness, and not to muscular tissue, much of which has disappeared. By pressing the electrodes deeply and using a strong faradic current, I get response in the anterior tibial and extensor longus digitorum muscles,—although she is not able to use these muscles,—but not in the other muscles below the knee. Knee jerk, muscle jerk, ankle clonus, and front tap are absent.

Testing for sensation, we find it is absent in the legs, and not until I reach about three inches above the umbilicus does she feel the points, and when we go backward up the spine the loss of sensation extends somewhat higher. She has, as most of these cases have, cystitis, an important matter which should be looked after. A deformity of the back can be seen about the ninth to the twelfth thoracic spines.

The symptoms, then, are those of motor and sensory paralysis to a certain height, total absence of tendon response in the lower extremities, loss of contractility in certain muscles, certain trophic conditions, and cystic paralysis and inflammation. Only two things could cause such a condition, either a lesion which crushed, or one which largely destroyed by inflammation her spinal cord, from about the eighth or ninth thoracic segments downward into the lumbar enlargement and conus, although it is probable that she has in fair condition some of the segments of the lumbar enlargement.

As the loss of sensation reaches to several inches above the umbilicus, it is probable that either by direct injury or by extension the lesion has involved the nerves as high as the seventh or eighth thoracic, at least. The sensory supply to about the line of the umbilicus is probably derived from the ninth or tenth thoracic nerves, and the region just above from one or two nerves higher. The eighth thoracic nerve passes out of the spinal cord about on a line with the beginning of the deformity.

Although the legs are totally paralyzed, loss of contractility and atrophy is not complete in them, several muscles below the knees escaping. It would seem as if this fracture had been one largely crushing the lower thoracic region of the cord, but to some extent affecting destructively, directly or by its consequences, the lumbo-sacral region. The upper limit of the lesion is best fixed by the highest level of the anæsthesia, which, as I have just indicated, would place it nearly as high as the origin of the exit of the eighth thoracic nerve, possibly as high as the seventh.

Cases similar to this have been worked out carefully by Thorburn and others. Usually the sensory and motor phenomena correspond to

nerves coming out a little below the injured vertebra, although we may have great variations in this respect because of irregularities in the primary injury or its inflammatory consequences. In some cases the spinal cord alone is crushed, in others the cord and the nerve-roots are involved in the injury. It would seem in this case that the anæsthesia was probably fully as high, if not higher, than the site of injury to the spine; an important practical point, as, according to Thorburn, in such a case we are in the presence of a lesion sufficiently severe to have compressed both the cord and its roots, and one, therefore, in which any operation would be utterly hopeless.

I was asked recently to see such a case at the Presbyterian Hospital, and I have been called to see other such cases after accidents. I have seen three or four operations in acute, and probably as many more in chronic, cases. Could this woman have been saved by an early operation? That is a difficult question to answer. My own experience would teach me not to operate in the very early stages; nor, on the other hand, is it well to wait too long. By early operation you are likely to do harm in cases where the indications are not clear. The probabilities are you will not succeed, though once in a while you may score a brilliant success. In this case it would probably have been best to have waited two or three weeks; then whether or not to operate should have been deliberately considered. She had some crushing of the cord and extravasation of blood, but not sufficient to kill her. The operator might at least have removed the counter-pressure, but of what avail would this have been if the cord and its nerves were completely destroyed? Every case of this kind should be gone over carefully.

FRACTURE AND HEMORRHAGE INTO THE CONUS AND CAUDA EQUINA.

CASE VI.—A. S., German, aged thirty-five, single, a silver-smith, on May 6, 1891, fell from the second story of a high building, alighting on his feet. He was unconscious for about half an hour, and when he regained his senses he suffered great pain in the back, about the lower dorsal region, and in the abdomen. His right wrist was fractured. During the first four weeks he was unable to move his legs, but after this he had some power in them. Involuntary twitchings and jumpings in them occurred at night. Two months after the accident he was put in a chair for several hours a day, but could not walk; he then had pain about the ankles, where the distress at times was extreme. From the first he lost control over his bladder and rectum, and for nine weeks he had to be catheterized. The bowels during this time

were moved only by means of enemata ; since they have been much improved, but even yet, if purgatives are administered, he may have constant involuntary discharges for several hours. The bladder is usually emptied by manual pressure over the hypogastrium, he being in the sitting posture. Urine dribbles away on slight exertion. Six months after the accident the patient could walk with the aid of crutches, and a little later without ; and then he got about fairly well, but his gait was stiff and he walked chiefly on his heels ; he felt as if he was treading on cotton. Four weeks after the accident consultation was held as to whether his spine should be opened or not, and from his condition now you see that it was well that it was not done. Remember that this man was totally paralyzed, yet he has no difficulty in walking now, although his gait is awkward ; one which shows there is something wrong with the muscles. He has gradually improved, and even yet appears to be gaining a little.

Let us study the movements of his lower limbs, starting above and being careful as to detail. I first have him cross his legs in a manner which should call into use his psoas and iliac muscles, as well as the adductors, and by having him place his foot over his opposite knee, the sartorius, etc., and in this way I shall go down the limb. He has all the movements above the knee, and below all fairly well, with a slight impairment of some, all showing rather more weakness in the left than in the right. He has some stiffness of gait, which is due to paresis or want of control over the muscles,—it seems to be in the gluteal group of abductors. When I ask him to extend and flex his toes, he does so, though, perhaps, with less power than normal. His toes are retracted and humped ; he can bend them up and down, but has some want of control over these movements, showing that the muscles supplying the toes are paretic, or that some old contracture is present.

I find partial anæsthesia on the dorsum of the foot and on the plantar surface, except in the area of the long saphenous nerve distribution. On the buttocks anæsthesia is absolute for several inches to either side of the median line, and downward from a line parallel with the coccyx to about the middle of the thigh, the area being much the same as that covered in sitting. He has sensation on the anterior part of the thighs, but none at the lower part of the scrotum, while it is present in the upper third of the scrotum. His penis is also anæsthetic. The anæsthesia defines certain nerve-supplies.

The following from a table modified by me, from one originally prepared by Starr, indicates the sensory supply affected in this case :

Fifth lumbar.—Back and outer side of leg, sole, dorsum of foot. (External popliteal, external saphenous, musculo-cutaneous, plantar.)

First and second sacral.—Back and outer side of leg, sole, dorsum of foot. (Same as fifth lumbar.)

Third, fourth, and fifth sacral.—Back of thigh, anus, perineum, external genitals. (Small sciatic, pudic, inferior hemorrhoidal, inferior pudendal.)

Fifth sacral and coccygeal.—Skin about the anus and coccyx. (Coccygeal.)

This man, then, has now left a lesion, which is chiefly interfering with the small sciatic nerves and their branches,—the inferior pudendal, pudic, inferior hemorrhoidal, and coccygeal nerves,—and those nerves which supply the bladder and rectum, which we now positively know come from the conus of the cord. Either a hemorrhage occurred in the cauda, or the conus has been compressed where the caudal nerves originate. He has imperfect anæsthesia in another area,—that which is supplied by the nerves which arise just above the small sciatic. The nerves of the lumbar plexus have escaped.

His reflexes are preserved. He has a slight deformity, which corresponds to the twelfth dorsal or the first lumbar vertebra.

Thorburn says truly that this cauda-equinal region is the safest place for spinal operation, as here you do not have the delicate spinal cord, but the same conditions as in operating on peripheral nerves. These nerves are not usually completely destroyed, as is often the delicate and highly organized region of the cornua of the lumbo-sacral cord. Still, as the cases often wonderfully improve, it is sometimes best to wait. If, guided by the slight deformity, the surgeon had cut downward and removed two or three laminæ and arches, no more benefit would probably have resulted than has come about through time. The fracture did not destroy the cord; more likely it caused injury to the caudal nerves and perhaps some hemorrhage into the cauda, which has been largely absorbed.

In two ways we may have hemorrhage into this region,—and I now speak of cases seen at autopsies and not from theoretical knowledge,—it may flow down from the seat of fracture and be entangled in the leash of nerves; later the blood may become absorbed, partly or wholly, giving a partial or nearly complete recovery as here. Occasionally in concussions of a grave character hemorrhage occurs in the substance of the conus or tip of the spinal cord. In a case seen with Dr. Willard, the man had a fracture about the eleventh dorsal vertebra; he was trephined and the bones removed, but he died. At the post-

mortem, absolutely independent of the bone injury, a hemorrhage had occurred in the highly vascular conus. I did not discover it at first ; not until I pulled apart the nerves of the cauda, when I found a little clot near the tip of the conus.

PROBABLE FRACTURE AND HEMORRHAGE INTO THE LUMBO-SACRAL REGION OF THE SPINAL CORD.

CASE VII.—The next case is probably suffering from a lesion in the lumbo-sacral cord. The notes, condensed, are as follows :

F. E., aged forty-one years, four years ago fell from a moving box-car, striking the lower part of his back. He did not lose consciousness but was unable to move his legs, and he felt as if the parts below the seat of injury, which were anæsthetic, were severed from the rest of his body. He was taken to a hospital, and for the first six weeks suffered excruciating pain in his back and legs, and was compelled to lie on one side. Since then he has had paroxysms of pain, but never

so severe. From the first he lost control over the bowels and rectum. He used a catheter for eighteen months, and now empties the bladder by abdominal pressure, having educated himself to void urine at regular intervals ; although if he laughs or makes extra exertion it passes involuntarily. His bowels are constipated and require large doses of cathartics to move them. On the back are evidences of old bed-sores. No marked wasting of the extremities is present. His feet are strongly extended. Small bruises on feet and legs are liable to become ugly sores.

The above notes were taken one year before the delivery of the lecture. Sensa-



Legs in a case of fracture and hemorrhage into the lumbo-sacral region of the spinal cord.

tion is now better, and rather more widely distributed on the anterior part of the thighs, just above the knees. He has considerable voluntary

movement in the right leg ; knee jerk is abolished, and no ankle clonus is present. Paralysis of the bladder continues. No active contractures of the legs are present. The feet are hyperextended somewhat, as in the foot drop of multiple neuritis. (Fig. 1.) The skin is blue and cold, and slightly glossy and œdematous. Faradic contractility is pretty well preserved in the right or unparalyzed leg, especially in the extensor quadriceps ; it is totally abolished in all the muscles of the left leg. A strong galvanic current gives the normal formula in the right quadriceps extensor. The muscles do not react on the right side below, nor on the left side either above or below the knee. He has occasional imperfect erections with ejaculation of semen, and with an imperfect voluptuous sense. Cremasteric movements of the testicles seem to be spontaneous and very sluggish. It is difficult to determine whether they are increased by irritation of the inside of the thighs.

A CASE OF CEREBRAL TUMOR.

CLINICAL LECTURE DELIVERED AT GUY'S HOSPITAL.

BY W. HALE WHITE, M.D., F.R.C.P.,

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London.

GENTLEMEN,—The case we will take for our lecture to-day is that of a little girl aged nine years who was brought to the Eye Department of the Hospital on December 8, for blindness. There is nothing in the family history to lead us to suspect syphilis or tubercle, and the child herself has always had good health till the commencement of the present illness. Three months ago the mother noticed that her daughter's eyesight was failing, and she thinks that for the last fortnight she has been quite blind. Two months ago, when coming out of school, she had a fit, which has been succeeded by others. After the fits she is sick, and complains of severe pain in the head. The fit begins with screaming, and the child puts her hand to her head on account of the severe pain there. All the limbs are thrown about and the eyes are rolled. The movements soon cease, then she has quiet for a few minutes. As she begins to recover consciousness she is sick at her stomach.

On admission.—She walked into the ward. She has no ataxia nor vertigo, but is obviously blind and cannot even see the light of a lamp. She is very thin and pale; the abdomen is remarkably retracted. The heart and lungs appear normal. There is nothing abnormal in the urine. Sensation is normal, and, although perhaps the legs are weak, there is no paralysis of any part of the body. The right knee-jerk cannot be obtained. The left is feeble; there is no ankle clonus. Plantar reflexes are absent, epigastric reflexes are present. The mental condition is variable. At times she is bright and cheerful, but she is generally quiet, and answers slowly and rather stupidly.

Eyes.—Both pupils are dilated, and neither reacts to light. Both disks are in a condition intermediate between neuritis and atrophy. The margin is a little indistinct; the disk is of a bluish-white color, and the arteries are small.

December 11.—The child had a fit. No one saw it, but afterwards she was sick, and cried because of headache.

December 26.—She has had several fits, in many of which there has been marked opisthotonos; she is always sick, and has much pain in the head after them. She also has headache apart from the fits. As the fits have gradually become more frequent, the mercury which she has had since admission has been omitted, and she has been given some bromide of potassium.

December 30.—During the last ten days the child has become markedly worse. She is thinner, and complains of more headache, especially at night. The fits are more frequent, for there are often three a day. She has been carefully watched to see if they begin in one part more than another, but this cannot be made out.

January 1.—She is very drowsy, and cannot feed herself, although she easily swallows when food is put into her mouth. The pulse has been frequently observed to be very slow, sometimes beating only forty to the minute. She has suffered much from constipation.

January 5.—There have been no fits the last few days. She is drowsy, and will not answer questions. The knee-jerk is present on the left side, but absent on the right. The wasting is very marked, and the abdomen is much retracted. The optic disks are much the same as on admission. The temperature is usually subnormal, often being as low as 96° F.

January 18.—She has gradually become more drowsy, and as she is now unable to swallow she is fed with nutrient enemata. The temperature went up suddenly yesterday to 103.6° F., and is at the same point this morning. The emaciation and the retraction of the abdomen are extreme, the anterior abdominal wall lying on the lumbar vertebræ.

January 19.—In much the same condition. The temperature is still high.

January 21.—She died this morning about 7.30 A.M. Half an hour previously the respirations were noticed to be failing, and the temperature rose to 106.4°. Sister says she noticed that the pulse went on beating for some time after the respiration had stopped.

This, then, gentlemen, was the case, and we have to diagnose it, and so you and I will go over the points that will help us to do so. The child was sent to us from the Eye Department, because, from the neuritis and atrophy of the disks, it was strongly suspected that she had a cerebral tumor; therefore it is our duty to examine this diagnosis to see whether it is probably correct.

Now, the symptoms of cerebral tumor are divisible into three groups. The first group contains those due to the mere presence of a foreign body within the cranial cavity. The second consists of symptoms due to the position of the tumor,—localizing symptoms, as they are called,—and the third group contains those due to the particular variety of tumor that is present, a rapidly-growing malignant tumor producing different symptoms from a slowly-growing innocent tumor.

First group. These symptoms are caused by the great increase of the intracranial pressure. If the tumor is large and rapidly-growing, it may of itself add considerably to the bulk of the cranial contents, but sometimes even very large tumors do not do this, because they destroy a sufficient amount of brain-substance to compensate for their own bulk. Cerebral tumors which either directly or indirectly impede the flow of blood through the veins of Galen also lead to great increase of the intracranial pressure, because of the collection of fluid in, and consequent distention of, the lateral and third ventricles which ensues upon pressure on these veins. This group of symptoms, due to increased intracranial pressure, contains the following :

(1) *Headache* due to the increased pressure jamming the meninges against the bone, and thus pressure is exerted upon the branches of the fifth and other nerves which supply the meninges. This headache, which is rarely or never absent in cerebral tumors, is often at first paroxysmal, but later on it becomes constant. You will remember it was so in our patient ; at first she had severe paroxysms of headache after the fits, later the headache was continually with her. It is usually very severe, and often keeps the patient awake at night. Both these points were also sometimes exemplified by our patient. Be especially careful in any case in which the headache keeps the sufferer awake at night, for then there is a very strong probability that it is not functional. The headache may be most intense over the tumor, but this is exceptional ; usually it is diffuse, as in our patient, for the increased pressure is felt equally in all directions. The pain is more frequently localized over the tumor in cerebellar tumors than others, because the cavity of the skull below the tentorium is a little cavity by itself, and therefore especially feels any increase of pressure within it. Occasionally when the tumor is near the surface of the brain there may be a tender spot on the skull. The most common mistake made about the headache of cerebral tumor is to conclude that because a patient has headache and difficulty of sight a cerebral tumor is present, when in reality both the symptoms are due to hypermetropia. I have known this mistake made, which, however, shows great carelessness. Always

be on the look-out for reflex headaches. I have known a headache thought to be cerebral cured by the extraction of a tooth.

(2) *Optic Neuritis*.—By far the commonest cause of optic neuritis is a cerebral tumor; the next most frequent is meningitis. So frequently is optic neuritis found in cerebral tumors that if it is well marked its presence should, if there are no signs of any other cause, always make us think of a cerebral tumor. I have, however, seen a case of primary optic neuritis which gave rise to the erroneous diagnosis of cerebral tumor, but primary optic neuritis is excessively rare. The size, locality, and nature of the tumor have no influence upon the degree or variety of the optic neuritis. The only fact in this connection that we know is that if, in a case of cerebral tumor, the optic neuritis exist in one eye only it will probably be on the side opposite to the tumor. Occasionally cases are met with in which the presence of a cerebral tumor does not give rise to optic neuritis, still the very necessary fact to remember is that it is of immense value in coming to a diagnosis of cerebral tumor.

It is extremely important also to bear in mind that the degree of loss of vision of which the patient complains bears no relationship to the degree of optic neuritis. There is at the present moment a woman in Miriam ward who can see very well, but her disks have already begun to atrophy. In certain exceptional cases the loss of sight may be quite sudden, but usually it supervenes gradually, as in the patient whose case we are discussing.

I am well aware that it is open to criticism for us to have included optic neuritis among the pressure symptoms, for some very eminent authorities, such as Dr. Hughlings Jackson, are strongly of opinion that it is not caused by increased intracranial pressure. Be that as it may, clinically it belongs to the group under consideration, for it is a symptom which, while indicative of the presence of a cerebral tumor, has no localizing value. It would take us too far afield to discuss all the theories that have been put forward to account for the presence of optic neuritis in cases of cerebral tumor. It will suffice if we state that the view that it is due to the influence of the increased intracranial pressure on the cavernous sinus or ophthalmic vein was shown to be wrong because the anastomosis between the orbital and facial veins is so free that pressure on the cavernous sinus does not lead to distention of the orbital veins. In very many cases the cavity of the sheath of the optic nerve, which is continuous with the subdural space within the cranium, is found distended when optic neuritis is present. This distention is doubtless due to the increased intracranial pressure, and the optic neu-

ritis has been ascribed to this distention. The objection to this view is that you may find extreme optic neuritis without any distention of the sheath of the optic nerve, even if a large cerebral tumor exists. A case we had in the post-mortem room on Wednesday in which, owing to the rupture of an aneurism at the base of the brain, hemorrhage had occurred into the subdural space, shows, as you can see, the optic sheath distended with blood, and thus demonstrates the continuity of the two spaces. Another widely-held view of the causation of the optic neuritis is that the tumor acts as an irritant foreign body, and causes an actual neuritis of the optic nerve within the cranial cavity, and this neuritis, by descending, causes the inflammation of the optic disk. One fact quite certain is that a descending neuritis in the optic nerve is often seen in microscopical sections when it never would have been suspected from the naked-eye appearances. I have often found this myself in sections, and have many which show it. On this view the tumor sets up a microscopic meningitis which implicates the optic nerve. This supposition explains those exceptional cases in which with a cerebral tumor the optic neuritis is present in one eye only. Perhaps the correct solution of the question is that sometimes the optic neuritis is due to increased pressure, and sometimes to a descending neuritis. Other theories are that of Hughlings Jackson, according to which optic neuritis is a reflex vaso-motor phenomenon due to the irritation of the tumor, and that of Leber and others who believe that in cases of cerebral tumor the fluid in the sheath of the optic nerve contains some particularly irritating material.

There is nothing especial to tell you in connection with the atrophy which in this case and most others follows upon severe optic neuritis. Mistakes in the diagnosis of the cause of optic atrophy can only be avoided by care in going over the whole of the case, and the causes of optic neuritis. I have known a case of supposed cerebral tumor turn out to be the optic neuritis of anæmia and the patient was cured by iron; on the other hand, I have known the neuritis caused by tumor put down as being due to anæmia.

(3) *Vomiting*.—Next to headache and optic neuritis, vomiting is the most common symptom of cerebral tumor. Often, if there are fits, as in our patient, it occurs in association with them. The characteristics of it are that it bears no relation to food; it is usually unattended with nausea, and commonly the tongue is clean. It may be paroxysmal. All these points are exhibited by our present case. Although in the greater number of cases it is caused by an increase of the intracranial pressure affecting the cerebral vomiting mechanism, still, some-

times when the tumor is in the posterior fossa it is due to direct pressure on the medulla.

(4) *Pulse*.—It is very characteristic of an intracranial lesion that the pulse should be slow. It fell to forty in our patient. The cause is that the increased pressure affects the intracranial cardiac mechanism.

(5) *Vertigo*.—This is a very common symptom of intracranial tumor. It may be due to the direct implication of the middle lobe of the cerebellum by the tumor, but usually is caused by the increased intracranial pressure.

(6) *Convulsions*.—You must always pay the greatest possible attention to these, for if they invariably begin in the muscles of some definite part, say the thumb or one toe, and from this gradually spread in the course of a few seconds to adjacent muscles, and if this march, as it is called, is always the same, and especially if the whole convulsion does not march far, so that say only one limb is affected, it is of the greatest possible value in diagnosis, for such a convulsion indicates a lesion in the cortical motor area corresponding to the convulsed part. You will remember that last winter, by means of such "Jacksonian convulsions" always beginning in the hand, we successfully diagnosed a tumor of the arm area of the cortex, and Mr. Lane excised it. But when, as in our present patient, the convulsions are general, and do not always begin in the same part, they are of no diagnostic value, and are simply due to increased intracranial pressure.

(7) *Intellectual Symptoms*.—You will hardly be surprised to hear that increased intracranial pressure commonly dulls the intellect, as it did in the present case. Ultimately the patient became stupid, drowsy, and comatose.

(8) *Enlargement of the Head*.—If the patient is of such an age that the cranial bones have not joined, the increase of the intracranial pressure will separate them, just as they are separated in a case of hydrocephalus, and the head will distinctly enlarge, and the veins on it become prominent. In the present case the child was too old for this to occur, but we thought that the roof of each orbit was driven down a little, so that the upper eyelid became more vertical than it is normally, a condition sometimes seen in hydrocephalus.

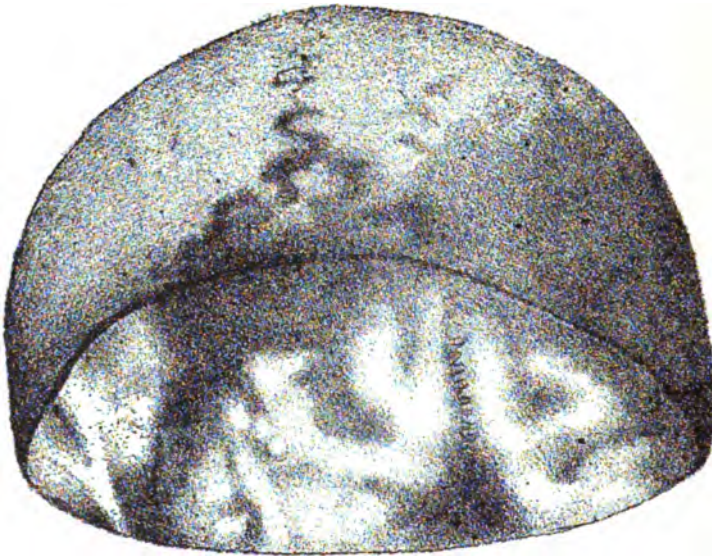
(9) *Wasting*.—Patients with a cerebral tumor usually waste extremely: our child was literally nothing but skin and bones. The cause of this is obscure; it certainly has no localizing value, and is probably due to the increased intracranial pressure modifying in some way the general nutrition.

(10) *Retraction of the Abdomen*.—This also is often met with in cases

of cerebral tumor. In our patient the anterior abdominal wall lay on the bodies of the vertebræ. Partly, no doubt, this is due, as the post-mortem here showed, to a total loss of all intra-abdominal fat, but this is not the whole explanation, for the retraction would not be so noticeable if the intestines contained their usual amount of gas. It is not of the slightest localizing value.

(11) *Constipation*.—This, as in our case, is one of the general symptoms of a cerebral tumor. The cause is not known.

(12) *Thinning of the Bones of the Skull*.—In some cases the increase



of the intracranial pressure is so enormous that the bones of the skull become very thin and atrophied, and this you must remember occurs when the tumor does not anywhere come in contact with the skull, and therefore it is simply due to the increased intracranial pressure. The bones become very thin, light, and transparent when held up to the light, which, owing to their tenuity, shows their vessels as a most beautiful arborescent net-work. The thickness, in extreme cases, of the bones of the vertex is no greater than that of very thin cardboard. The specimen I have here shows all these points admirably. (See Figure.) It was taken from a case of cerebral tumor. We have a specimen in the museum in which this thinning from increased intracranial pressure has actually laid open the tympanum. There is nothing noteworthy about the exterior of the skull, but the inner surface of the

bone is roughened like sand-paper. Sometimes this thinning is so marked that it can be detected during life, for the bone can be pressed in, when it rebounds like the bottom of an oil-can.

(13) *Temperature*.—You know that sometimes this is of localizing value, the best-known instance being the rise of temperature that you may find associated with a tumor of the pons. Apart, however, from this question we occasionally notice that, as in the present case, shortly before death there is a great rise of temperature. In our patient it rose to 106.4° F. I know of cases in which it has risen to 107.4° and 108° F. Such an extreme rise is of great prognostic value, for it means that the end is near. It is of no localizing value, for it is probably due to the fact that the increased intracranial pressure has at last exhausted the cerebral thermic mechanism.

These general symptoms, gentlemen, are those which indicate the fact that your patient has a cerebral tumor without helping you in the slightest to the position of it. I have given you them in the order of their importance and frequency. You will all agree that we were able from them easily to say that our patient had a cerebral tumor.

The second group of symptoms indicates where the tumor is. They are the localizing symptoms, and owe their presence to the fact that the tumor locally destroys some part of the brain, which has a local definite function. For example, if the patient had paralysis of the third nerve we might conclude that there was a tumor at the base of the brain pressing on it. We had an instance last week in which an intracranial aneurism pressed on the third nerve and caused paralysis of it. If the tumor causes local fits in one arm, as in the woman we had in a year ago, it means that it implicates the cortical arm area on the opposite side. If the tumor causes glosso-labio-laryngeal paralysis it is somewhere about the medulla. If it causes ataxia it implicates the middle lobe of the cerebellum. If it gives rise to contracted pupils and crossed paralysis it is in the pons. Now, in our case the tumor caused absolutely no localizing symptoms. In what places, therefore, can a tumor be and yet cause no localizing symptoms?

By far the most frequent seat for it in such a case is either lateral lobe of the cerebellum or either frontal lobe. These are not known to have any localizing value. A tumor in the occipital lobe or in the path of visual impulses might, conceivably, when there was optic atrophy, cause no localizing symptoms, for the presence of the atrophy would render it impossible to detect the amblyopia or hemiopia that should result. I show you here a large tumor of the cerebellum which gave rise to absolutely no localizing symptoms, and I have met with

several instances of tumor of the frontal lobe causing no localizing symptoms. Perhaps one of the most striking was a patient, the sister of the waiter at Dr. Moxon's club, who complained only of slight headache, and whom Dr. Moxon would not have taken into the hospital except to oblige her brother. Optic neuritis was found and cerebral tumor diagnosed. She was intelligent, and saw perfectly. Before admission she had been arranging about furnishing her house, as she was about to be married; when in the hospital she helped the nurses in their work. One day she dropped down dead, and a huge glioma was found in the left front lobe. The probable reason why tumors of the frontal and lateral cerebellar lobes cause no symptoms is that these parts are concerned in intellectual operations, and therefore their functions are less fixed in local parts than are the functions of other parts of the brain, for the intellectual functions have been later developed both in the evolution of the species and in the life of the individual, and it is well known that a long period of evolution and increasing age of the individual are both associated with a localization of function in definite parts. As the functions of the frontal and lateral cerebellar lobes are feebly fixed, when these parts are destroyed neighboring parts or possibly the corresponding part of the opposite side of the brain will probably be able to take on the functions of the destroyed parts.

The outcome of our diagnosis so far is that our patient has a tumor of the brain in some part which does not give localizing symptoms; probably, therefore, in the lateral lobe of the cerebellum or the frontal lobe.

The third group of symptoms we have to consider are those which indicate what the tumor is. Out of one hundred consecutive cases of cerebral tumor that have occurred in the post-mortem theatre of this hospital the growth was tubercular in forty-five, a glioma in twenty-four, a sarcoma in ten, a carcinoma in five, a gliosarcoma in two, a cyst in four, a gumma in five, a lymphoma in one, a myxoma in one, and doubtful in three. There was in the present case no evidence of growth elsewhere in the body, so it was almost certain that the cerebral tumor was not a sarcoma nor a carcinoma. We were last year able to diagnose a sarcoma of the brain from the fact that the patient had had one in her breast. There was no evidence of congenital syphilis in the present case, so a gumma was out of the question, and, therefore, we were narrowed down to a diagnosis between a tubercular tumor and a glioma, for the others are so very rare. Now, of the above forty-five cases of tubercle, twenty-four, or more than half, were under ten years of age, but of the twenty-four gliomata only two were under ten years of age, so that out

of twenty-six cases of tubercle and gliomata under ten, only two—that is to say, about eight per cent.—would be gliomata; therefore there was a very strong likelihood that our patient, who was only nine, was suffering from a tubercular tumor. You learn from these figures the great probability that any cerebral tumor in children is tubercular. In this particular case the probability that the tumor was tubercular was enhanced by the fact that we had reason to believe it was in the cerebellum, for that is a very common seat for a tubercular tumor. The only reason against this view was that there was no evidence of tubercle elsewhere in the body, and this was strongly against it, for tubercle of the brain is almost always associated with tubercle elsewhere, still the tubercular disease elsewhere often gives no symptoms.

Autopsy.—Now I show you the brain, and you will see that our diagnosis was partly correct; there is a cerebral tumor growing in a position where it would give no symptoms, for it is a growth, the size of a filbert-nut, springing from the right optic nerve just anterior to the optic commissure, pressing on the left optic nerve and growing upward through the anterior perforated space into the third ventricle, where you see it bulging. The upper part of it is cystic. By its pressure backward it had doubtless greatly impeded the flow through the veins of Galen, for both lateral ventricles were much distended, and the convolutions of the brain were flattened, thus explaining the pressure symptoms. There was no sign of meningitis. As the patient was blind when she came in, we could hardly have been expected to diagnose a tumor in such a very unusual position. The principle of our diagnosis was correct,—namely, that the tumor was not producing localizing symptoms. It certainly is not tubercular; in all probability, histologically, it will turn out to be a glioma. The figures I have quoted show how excessively rare such a tumor is at such an age. Perhaps we ought, considering the absence of signs of tubercle in other organs, to have been more chary of expecting the growth here to be tubercular.

Prognosis.—This in all cases of cerebral tumor is very grave, but possibly certain rare cases recover. Here is a child who came into the hospital in 1885, and was under my care. She then had optic atrophy and headache but no localizing symptoms whatever. She has now recovered from the headache, and is well except that she is quite blind; she is very intelligent and can play and sing. Probably her tumor is in the lateral lobe of the cerebellum or the frontal lobe, is tubercular, and has become quiescent. I have seen one other patient recover. In that case the tumor was in the frontal lobe and implicated the frontal

bone through which it grew. The opening was enlarged and the tumor was burnt away with nitric acid. The child made a complete recovery, and gained prizes at board schools. But these cases are so rare that they should hardly influence you in your prognosis.

Treatment.—What we have to say about this is really a part of the subject of prognosis. If there is a fair amount of evidence that the tumor is a gumma, there is some hope, for you may absorb it by means of full treatment with iodide of potassium and perchloride of mercury, but remember that only five per cent. of all cerebral tumors are gummata, and that by the time you get the case the gumma may have done irreparable and fatal damage to the brain, and that even if you absorb it the scar tissue left may cause serious symptoms. Some tumors may be cut out, but unfortunately the great majority are either too difficult of exact localization, too diffuse, too large, out of reach, multiple, or associated with disease elsewhere in the body. Judging from my own experience I should say that probably not much more than ten per cent. of the patients are capable of being relieved by operation, although in proportion as the cases are recognized early so will this percentage increase. If you decide that the case is not one for operation, and the one we are discussing certainly was not, as we could not localize the tumor, you should either give cod-liver oil, if you think it is tubercular, or else large doses of iodide of potassium and mercury in the hope that it is syphilitic. I usually give an eighth of a grain of the perchloride of mercury in five minims of water injected deep into the gluteal muscles once a day, and thirty grains of iodide of potassium thrice a day; this gets the patient under treatment rapidly, and this is all-important.

Mode of Death.—Under this heading I wish to direct your attention to two strange facts. One is the number of cases of cerebral tumor which died suddenly. In fourteen out of thirty-one cases death was sudden. For instance, in one the nurse in the middle of the night lifted a patient with a cerebral tumor forward to give him something to drink; he fell back dead. A woman was standing peeling some potatoes; she dropped down dead. One patient was noticed to be all right; a few minutes after when the nurse went to the bed he was dead. Probably the reason of this sudden death is that some slight shifting of the head brings the increased pressure to bear especially on some vital part, most likely the vagi.

The other fact is that often the pulse goes on for quite a long time, it may be half an hour, after the breathing has stopped. That was so in our present case, and I know of several others. No satisfactory explanation has, as far as I know, ever been given of this. Strange to

say, the patients do not turn livid, although the respiration stops completely. I never heard of a case in which the pulse stopped first and the respiration continued.

Etiology.—It is a well-known fact that three males have tumors of the brain to one female. This has been stated to be because men are much more liable to blows on the head than women, for in some cases it appears that the tumor has developed after a severe blow. Our figures at Guy's, however, show that this is not the sole explanation, for male children are more liable to tumors of the brain than female children. The cause of this predisposition of the male sex is quite unknown.

LESIONS OF THE MOTOR TRACT.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC.

BY B. SACHS, M.D.,

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GENTLEMEN,—Let us consider to-day the symptomatology of lesions of the different parts of the motor tract. The motor tract is that part of the central and peripheral nervous system concerned in the transmission of motor impulses from the cortex to the periphery. Interruption of the motor tract in any portion causes loss of motion or paralysis. The motor tract has its origin in the motor area of the hemisphere. This area is located about the fissure of Rolando, in the præcentral and postcentral convolutions, which we can further subdivide, as on the chart before you, into centres governing motion in the leg, arm, face, and organs of speech. By this we mean that excitation of each special portion most easily produces motion in the segment which it controls upon the opposite side of the body. From the cortex the fibres of the motor tract pass down through the hemisphere into the region of the basal ganglia. In the subcortical portion of the hemisphere the motor tract forms but a small part of the white fibres of the brain. We find in addition sensory tracts and the commissural fibres or those which connect one portion of the cortex with another, and one hemisphere with the other. In connecting the parts above with those below, the motor tract actually passes through the great ganglia, occupying a space between the nucleus caudatus and the lenticular nucleus, and between the latter and the optic thalamus. This region is spoken of as the internal capsule. The internal capsule is largely occupied by the motor tract. Here the fibres are closely massed together, diverge within the centrum semiovale, then towards the cortex like the ribs of an umbrella. From the internal capsule the motor tract passes through the crus, lying on its ventral aspect, and passes on successively through

the pons and medulla, where the most of the fibres cross over and pass down through the lateral column in the opposite side of the cord. A small portion pass down on the same side in the anterior column of the cord. In the crus, pons, and medulla the fibres are in juxtaposition with the roots of the cranial nerves. These facts furnish us with our basis for the localization of lesions.

In the spinal cord fibres of the motor tract are given off at each level for the parts in relation with that special level. Consequently the tract dwindles in size; it becomes smaller as we descend in the cord. In the cervical region fibres are given off to the arm; in the dorsal region to the intercostal and abdominal muscles, and in the lumbar region to the lower extremities.

For diagnostic purposes the motor tract may be broadly divided into two divisions: that from the cortex to (but not including) the anterior horns of the cord, and that from the anterior horns to the periphery. Lesions in each of these divisions have certain common and broadly distinctive symptoms. The general symptoms of lesions in the first division are,—

1. Hemiplegic form of paralysis.
2. Exaggerated reflexes.
3. Spastic contractures.
4. Absence of muscular atrophy.
5. Absence of change in the electric reaction.

Generally speaking, these symptoms characterize lesions of this division whether in the cortex, internal capsule, pons, medulla, or lateral columns of the cord.

The symptoms of lesions of the second division are,—

1. Paralysis of irregular distribution, often bilateral.
2. Diminution or loss of reflexes, flaccidity of the muscles.
3. Distinct muscular atrophy.
4. Changes of the electrical reaction: either simple diminution or some form of the reaction of degeneration.

To aid us in remembering these distinctive symptoms, we should bear in mind the functions of the ganglion cells of the anterior horn. These cells control the tonus of the muscle, they preside over its nutritive processes, and preserve its normal electrical reaction. In lesions of the peripheral portion of the motor tract, the influence of the anterior horn cells upon the muscle is lost; in lesions of the central portion it is not. It is thus easy enough to decide whether a lesion is above or below the anterior horn; and, roughly speaking, cases of paralysis may be divided into brain palsies and spino-peripheral palsies.

BRAIN PALSIES.

We differentiate brain palsies still further, according to the location of the lesion.

Cortex.—If the lesion is cortical we have the general symptoms of brain palsies, modified by the fact that the paralysis is not necessarily hemiplegic. The cortical centres are so wide apart, and their areas are of such dimensions, that a lesion may involve a single area only, and, instead of hemiplegia, monoplegia will be the result. Our lesion may, on the other hand, be large enough to cover all three centres, and we may have hemiplegia even from a cortical lesion. This is especially true of paralysis from meningeal hemorrhages in children. As a rule, however, cortical hemorrhages are less extensive and often produce paralysis of only an arm and a leg, of the face, or of the speech organs. Tumors, as a rule, paralyze only one part,—as the leg, arm, or face. If this paralysis is limited in extent, however, there are still present the other general symptoms,—the increased reflexes, the contractures, and the absence of atrophy. Having these symptoms with paralysis of limited extent, we know that the lesion is in the cortex. Limited paralysis should, therefore, make one suspicious of cortical lesions. We can get absolute monoplegias from lesions nowhere else in the brain.

The cortex is par excellence the starting-place for convulsive movements. Localized epilepsy is a valuable corroborative symptom of cortical lesion. Repeated convulsions are especially significant. We may have an initial general convulsion from a lesion in the internal capsule, the irritation being carried to the cortex; but this convulsion is not apt to be repeated. Disturbed mentality is also a feature of cortical impairment. Coma and delirium are more frequent in lesions of cortex than in lesions of other parts of the brain. Added to these distinctive marks slight sensory disturbance also may be present.

Internal Capsule.—Lesions of the internal capsule are distinguished from cortical lesions by the fact that they give rise to hemiplegia, pure and simple, without marked sensory symptoms. They never give rise to monoplegia, nor to any implication of the cranial nerves. The fibres are so massed together that no lesion is so small as to affect only those going to a single part. There may be an initial convulsion, but this is rarely repeated. There is initial coma alone, unless the lesion is so extensive as to take in neighboring parts of the brain.

Crus, Pons, and Medulla.—In lesions of the motor tract in these regions, we have hemiplegia with involvement of the different cranial

nerves; the special nerves involved varying with the location of the lesion. If the lesion is in the crus we have hemiplegia of the opposite side of the body, with paralysis of the oculo-motor muscles of the same side. Crossed third-nerve paralysis is a feature of lesions in this location. The fourth nerve may be also involved. In pons lesions we have involvement of the seventh, sixth, fifth, and eighth nerves, this being the order of frequency in which they are affected. In lesions of the medulla the ninth, tenth, eleventh, and twelfth nerves may be affected. In the pons and medulla the motor tracts lie so close together that we may have either hemiplegia or diplegia; the latter even from a small lesion, with complete paralysis of both arms and legs. Lesions in the upper part of the pons, above the origin of the facial fibres, may closely resemble internal capsule lesions.

The infantile cases presented in this clinic have, as a rule, cortical lesions; they have initial and repeated convulsions; the arm is chiefly affected, the leg less markedly. There is no marked atrophy and no change in the electrical reaction.

In the ordinary case of hemiplegia in the adult, the lesion is in the internal capsule; there is an initial convulsion, coma of a few hours' duration, paralysis with few or no sensory symptoms, contractures, increased reflexes, and no change in the electrical reaction.

A lesion of the crus was represented here by the case of a woman with complete paralysis of the right side. The eye of the opposite side showed ptosis, the lid dropped and the eye was abducted. There was an outward pull, showing that the muscles which antagonize the external rectus were paralyzed. It was apparent that all the muscles supplied by the third nerve were affected. Autopsy showed thrombosis of a branch of the posterior cerebral artery.

In the case of a man we found complete hemiplegia of one side with paralysis of the opposite side of the face and marked sensory disturbance of the face, the facial nerve and the trigeminus were involved. The man suffered from syphilitic lesion of the pons.

In another case of hemiplegia there was marked hemiatrophy of the tongue upon the same side. The hemiplegia soon changed to diplegia. From this condition he recovered gradually, and finally paralysis of one arm and atrophy of the tongue remained, and he is now recovering even from this atrophy. In this case there was undoubtedly a lesion of the base, involving the medulla chiefly. The disease spread from one-half of the medulla to the other. We must assume a lesion of this kind or multiple lesions, and the latter are never to be assumed where a single lesion will explain the symptoms.

It will be seen that the most important distinctions are those by which we distinguish cortical brain lesions from others, as it is in the former cases that operative interference is attempted. We do not yet try to penetrate to the base of the brain, and only one or two very bold operators have sought a lesion in the internal capsule. We want, then, particularly to remember the distinctive marks of a cortical lesion. These are monoplegia or hemiplegia, repeated localized convulsions as well as coma and delirium. There may be slight sensory disturbance. These distinctive signs, when combined with contractures, increased reflexes, absence of atrophy, and absence of the reaction of degeneration, determine the diagnosis of a cortical lesion. We may have to differentiate such a cortical lesion in a case like the following: A child is born after a difficult labor; a few days later the child is found to be paralyzed in one arm; you want to determine whether the origin of the paralysis is in the brain or whether the brachial plexus simply has been injured. In both conditions you have monoplegia; but if the monoplegia is of central origin, you will have repeated convulsions and spastic paralysis; while, if the monoplegia is of brachial origin, you will have flaccid muscles, changes in electrical reactions, and marked atrophy within a few days. All the reflexes will be abolished. The distinctions between cortical and peripheral palsies are easily recognized. In our next lecture we shall go on to consider the signs by which we distinguish spinal and peripheral lesions from one another.

ATAXIC PARAPLEGIA.

CLINICAL LECTURE DELIVERED AT THE GENERAL HOSPITAL.

BY ROBERT SAUNDBY, M.D., F.R.C.P.,

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GENTLEMEN,—Ataxic paraplegia, or combined lateral and posterior sclerosis, is a disease which presents a combination of the symptoms of posterior and lateral sclerosis. This was called ataxic paraplegia by Dr. Gowers, and, though not altogether an appropriate name, it is a convenient one.

The patient before you, aged forty-six, was admitted on December 19, 1893, complaining of pain in the legs and lower part of the back, with loss of power in his lower extremities and failure of eyesight. These symptoms had been coming on for about a year. He is, as you can see for yourselves, a small, dark man, ill-nourished, but not anæmic. He is very deaf and nearly blind, and these sensory failings give to his face a dull and stupid expression that disappears when he hears what is said to him, especially if he finds that he is also understood. He tells us that eighteen months ago he had a sense of constriction round his waist, "as if somebody was fastening a belt tightly around him," a symptom of myelitis, which is known as "girdle-pain." Soon after this he noticed that his eyesight began to fail, his legs became stiff, and his "knees came together" so that he walked with difficulty and soon got tired. His gait gradually grew more uncertain, especially in the dark. He suffered from rheumatoid aching in the knees and ankles, and darting pains in the backs of the thighs; these were worse while he was in bed. He has frequently had cramps in the abdomen, and lately lumbar and sacral pains. On inquiring into his family history it appears that one of his sisters died in Stafford Asylum, where she was sent for melancholia and suicidal attempts, but in other respects it presents no facts of interest. He has never been married, and, although his work as a laborer exposed him to the weather,

he has enjoyed general good health. His deafness dates from boyhood; both ears discharge frequently, and both membranes are perforated. Twenty years ago he had syphilis, and his habits have not always been temperate, but for the last fourteen years he has been an abstainer. He has never met with any serious accident or injury. There is no impairment of his mental functions, although his pronounced deafness makes him appear stupid, his memory and intelligence are quite normal. If we ask him to stand up he can do so with difficulty, but he stands steadily and does not fall when he shuts his eyes. He takes very short shuffling steps and walks on his toes, dragging his feet and crossing them over one another, so that his progression is most unsteady and insecure. He turns with great difficulty. On examining his lower extremities the muscles are found to be firm, and not tender on pressure; the thighs are adducted and the knees extended; when bent they give way with that peculiar sensation which has been aptly compared to the bending of a leaden pipe. Neither ankle-clonus nor knee-jerk can be obtained on either side, and the superficial reflexes are also absent, but he can feel a tickling in the soles of his feet very plainly. There is no want of co-ordination in the movements of the upper extremities, which are apparently quite unaffected by the disease; in the lower limbs movements are much impaired, owing to the spastic condition of the muscles; but on looking at his bare feet we do not see any irregular or ataxic action of the extensor tendons. Cutaneous sensibility seems everywhere normal; there is no bladder or rectal trouble; and all the muscles respond well to galvanism. He has slight convergent strabismus, due to spasm of the right internal rectus; the pupils are dilated and do not react to light. There is some nystagmus on turning the eyes strongly outward. The field of vision is diminished in the left eye, and both optic disks are pale, with blurred margins, especially the left, the result of atrophy following neuritis. I need not trouble you with a detailed account of the state of the other organs, but at the apex of the heart there is a slight systolic murmur. Since admission he has passed the greater part of his time asleep, but he has been so unfortunate as to suffer in succession from acute catarrhal pharyngitis, acute gout in the right ankle, and intense catarrhal conjunctivitis in the right eye. He is at present recovering from the conjunctivitis.

This case presents the following symptoms of posterior sclerosis (tabes dorsalis):

1. Westphal's sign, or loss of knee-jerk.
2. Argyll Robertson's sign, or loss of the pupillary reflex to light.

3. Lightning pains.
4. Girdle-pain.
5. Atrophy of the optic nerves.

We may note the absence of,—

1. Ataxic movements.
2. Romberg's sign, or inability to stand with the eyes shut.
3. Visceral crises.

Depending on the sclerosis of the antero-lateral columns, we recognize,—

1. Spastic paraplegia of the lower extremities.
2. Nystagmus.

While we are struck by the absence of,—

1. Exaggerated knee-jerks.
2. Ankle-clonus.

Let us briefly summarize the essential facts in this case. In the history there is evidence of neurotic heredity, a sister having been insane; he has had syphilis twenty years ago, and he admits having drunk heavily of both beer and spirits. The first indication of his present illness was girdle-pain, eighteen months ago, and this was followed by failure of eyesight, stiffness and adduction of the thighs, lightning pains, and increasing loss of power in the lower limbs. If we can credit this history, it points to a chronic diffuse myelitis associated with optic neuritis, and followed by sclerosis of the antero-lateral and posterior columns, which seems to have run an equal and parallel course.

We are very much in the dark about the origin of these forms of sclerosis. We can recognize that the lesion affects certain regions of the cord, as physiology gives us the key to the relation between certain lesions and certain definite symptoms; thus we understand how a lesion of the posterior columns intercepts the centripetal paths of sensory impulses going to centres in the cord and brain, abolishes the deep reflexes, and may cause ataxic movements with sensory anæsthesia. We understand, too, how sclerosis of the antero-lateral columns interferes with the efferent motor impulses in the pyramidal tracts, causing muscular paresis, and we are familiar with the fact, though we cannot explain it, that descending degeneration of nerve-fibres leads ultimately to rigidity in the paralyzed muscles. But we do not know the starting-point of the disease, or why it should commence in, and confine itself to, well-defined and very limited symmetrical portions of the spinal cord, nor why, in certain other examples, like the one we are considering, we should have a combined lesion still limited and symmetrical, but affecting both sensory and motor tracts. Some of these cases commence in

a definite attack of myelitis or spinal hemorrhage, but in most there is no recognizable primary lesion, and no cause, either direct or indirect, can be discovered.

You probably know that syphilis is regarded as the principal cause of tabes, and its relation to some cases of primary lateral sclerosis is admitted; but in ataxic paraplegia no such frequent relation has been observed. How does syphilis cause sclerosis? Certainly not by a primary gummatous deposit degenerating into fibrous tissue. If this were true the problem would be simple, and there would be no doubt of the true relation. Unfortunately, it cannot be claimed that this is the usual mode in which sclerosis starts, and we must admit our ignorance of the exact manner in which syphilis acts in determining these changes. In the present case there is a history of syphilis, and we may content ourselves with noting the association without speculating as to whether the connection is causal or not. We may also note the tendency to nervous disease in the family, of which his sister's lunacy affords evidence. Here again we touch upon a very obscure subject of which we really know nothing, though the analogy of proper soils for plants, or of suitable media for the cultivation of bacteria, suggests a conception of the nature of the relation which we have no warrant for accepting as true. However this may be, experience proves the enormous importance of family predisposition for the development of diseases, by no means only of those known to be due to micro-organisms or even of those supposed to be "infective" in origin. Whether the exposure to cold which his occupation entailed was the immediate exciting cause, and his neurotic heredity the predisposing cause, cannot be dogmatically affirmed, but such a view of the etiology of the case is not unreasonable.

The symptoms exhibited by this patient are in part those of locomotor ataxia and in part those of spastic paraplegia, but they are not combined quite typically.

To justify the name of ataxic paraplegia there should be, and usually is, ataxic movement, but this symptom was absent. Spastic paraplegia is generally associated with increased knee-jerk and ankle-clonus, neither of which was present. We know too little about this disease to attempt to say why in a given case there should be this or that special distribution of symptoms; but it is not unnatural that sometimes these incline more towards the ataxic and sometimes towards the spastic type, and that endless combinations of the two groups should present themselves. We cannot, therefore, affect any surprise that this case does not conform in every respect to the descriptions given in the text-books.

The prognosis of this particular case is unfavorable for recovery, as the disease is in a very advanced stage, and before long the poor fellow will be unable to get about. Since his admission his power of walking has sensibly diminished. His life may last a long time, but this will depend on circumstances. He will probably gravitate to the workhouse infirmary, where he will be well cared for and have the best chance of living. Treatment cannot, unfortunately, be of much direct use, but we must do what we can. He was treated on admission by the inunction of mercury, a drachm of blue ointment being rubbed into his axilla every night. But his intercurrent troubles have interfered with this treatment. The chief means indicated are,—

1. Rest, especially useful when there is marked ataxy.
2. Hot-air baths and upward rubbing of the muscles, to soften them and relieve the spasm. For internal remedies, mercury and iodide of potassium, arsenic, iron or quinine as general nervine tonics, and antipyrin or phenacetin to relieve neuralgic pains when these are present. To such or similar measures we may wisely limit our therapeutic interference.

PARANOIA, OR DELUSIONAL INSANITY.

CLINICAL LECTURE DELIVERED AT THE VANDERBILT CLINIC.

BY M. ALLEN STARR, M.D., Ph.D.,

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GENTLEMEN,—Let us question this patient and endeavor to find out what is the matter with him.

Q. The doctor tells me that something has been troubling you of late? A. Well, it has for some years back.

Q. How long ago did your trouble first begin? A. About two years ago.

Q. What was it that first attracted your attention? A. I was pursued and I heard voices.

Q. Were they voices that you recognized, or were they strange voices? A. Sometimes a man's and sometimes a woman's.

Q. Did these voices say the same thing, or say different things? A. That was what I wanted to explain to you. These voices had much gift of gab and gave me much information in regard to keeping myself in good health. The first I heard of it was that I had a heavy cold and they told me how to treat myself, and I can give you the treatment now.

Q. Were they common-sense things—things that you would have thought of yourself? A. Yes.

Q. Did they call you names? A. Yes, more than there are in the dictionary.

Q. Do they trouble you in the night as well as in the day? A. Every minute that I am awake; and they can tell my very dreams and thoughts.

Q. Do you hear them distinctly? A. They act so on my mind that I can hear almost everything.

Q. Are these things offensive to you? A. They are not now, but there was a time when I thought it was due to human beings pursuing

me; but as soon as I found it was done by instruments and by men operating them, I didn't mind it.

Q. What kind of instruments did they use? A. I can't tell. They call them speaking tubes, and they say they are men belonging to the Academy of Natural Sciences and they have selected me for a subject.

Q. So they act upon you from a distance through these tubes? A. Yes, no matter where I go. On the horse-cars, the steam-cars, in church, everywhere.

Q. You are sure you hear them as you hear me? A. I am.

Q. Have they ever injured you in this way? A. Well, they claim they have hurt me by making people believe that I am crazy. People know of this and say I am crazy, and they tell people to get away, saying that I will kill them.

Q. Have you been at work all this time? A. I have been studying, and have travelled looking for work.

Q. Do you think they have conspired against you to prevent your getting work? A. They say they have, and I have had to leave my family and look for work somewhere else.

Q. The Academy of Natural Sciences meets in different cities, does it not? A. I suppose they have branches.

Q. They met the last time in Philadelphia. Were you there at the time? A. I was not.

Q. Did you take notice of their meeting? When they are meeting are you affected more than at any other times? A. I have not noticed it.

Q. As far as your general health goes, they have not succeeded in affecting that, have they? A. They have by their suggestions done me good.

Q. So it is not all evil that you have heard? A. I will tell you all about it. There is not one word from me from the day I was born that has not been hurled back at me. Every person, man, woman, or child, that I have met in my travels has noticed me. There was a time, before I found that this was done by instruments, that I believed that I was really on the brink of insanity, because I did not sleep at nights—sometimes only two or three hours. I used to lie awake and listen to these speaking-tubes.

Q. Did they depress you? A. Certainly. Everything that I did, good, bad, or indifferent, was being hurled at me.

Q. It could not have been your own thoughts that were so vivid that you almost heard yourself think? A. No; because every word

that you have spoken to me will be hurled back at me, and the description of this hall and these gentlemen here.

Q. Is your general health at present good? *A.* It is very good.

Q. You have no disease at all,—no ringing in your ears besides these voices? *A.* No, sir; my hearing is benefited, I think.

Q. Your sight is good? *A.* Yes.

Q. That is a society of able men, and they are not likely to do anything to injure you in any way. I think the fact that they have advised you about your health shows that they are disposed kindly towards you. They have never done you any harm, have they? *A.* I was run down and didn't sleep much, and I knew exactly what was the cause of it. I lost my appetite and then they gave me ideas to restore myself to health again. I used to have sick headaches quite often, but do not any more, because they have given me a remedy to prevent them.

Q. Tell me what it is. *A.* I used to have a sick headache, I suppose, once every two weeks, and sometimes once a week. Sometimes I might eat a lunch which would cause a sour stomach and give me a sick headache. They told me to heat water in the mornings and after it boils to let it cool so that I can drink it without scalding myself. They explained to me that when you take hot water and pour acid on it, there is an explosion just like touching a match to gunpowder. Well, after sleeping all night there is an accumulation of bile in the stomach, and that is acid; and when you drink the hot water down—not sip it—it causes an explosion.

Q. Do you feel that? *A.* I can't help feeling it inside of five or ten minutes, for I belch up wind just as you do when you drink any effervescing water. I raise wind and then sour phlegm, because this hot water makes an explosion and scatters it; and after that takes place I raise the phlegm, and that is what causes sickness and causes a man to be sick all day long, because he has the phlegm there.

Q. That is a very good prescription for sick headaches. *A.* People who drink hot water don't know how to drink it.

Q. Do you think that there is anything that you could take internally that would in any way affect the sound of these voices? You say your hearing is rather acute, do you think anything would affect it? *A.* No, I don't think it would. They say, one day when they can't accomplish their purpose they will give it up.

Q. I will talk to your doctor about you, and if it is possible we will stop this.

[The patient leaves the room.]

Gentlemen, I brought in that case because in our course we shall

soon begin to study these cases of mental disturbance. Now, that is a case that is not at all uncommon, and is an example of a disease known as paranoia. Paranoia is a new term for the condition that has been long known as monomania, or delusional insanity, and is just the condition that you find in many celebrated criminals. Guiteau had this disease, and so did the man who shot at Emperor William. In going back through history we find that very many of the crowned heads of Europe have been at various times shot at or had other attempts made on their lives by persons suffering from this disease. I want you to remember the case, and to remember certain features about it. I had him to face you while speaking, so that you could notice the play of his features. I hope you noticed the continued tremor of all the finer muscles of the face, the forehead, and his eyes and eyebrows. This is not at all characteristic, as it is in paresis; it is only an indication of his nervous agitation. I think you probably noticed once or twice, while I was talking to him, a sudden turning and looking in a different direction. At those moments I think he heard the voices coming from that corner of the room. From little indications of that kind you can often judge of the occurrence of hallucinations. His delusion is one of persecution, and is perfectly logical. His way of reasoning is as correct as yours and mine. The trouble lies not in the process of reasoning, but in the premise with which he starts. Suppose that he hears these voices, he can reason out the explanation of them. We all try to get an explanation of anything that we see and hear, and his is simply a reasonable inference from the premise with which he starts. This is a point to be considered in cases of this kind. In this man, fortunately, some of these hallucinations have been in the line of things favorable to him. Of course, some one has told him that hot water did good in the morning, and in his condition it did do him good. If from repeated conversations with such a man you find he has no special feeling of animosity, you can allow him to go about; but if you find—and it is important to try to find out—that the patient is cherishing a feeling of resentment against any person or set of people, that is the kind of lunatic that should be put into an asylum and kept there. They are very hard patients to keep there, because their minds are very keen, and they are the very kind that will use every available means of getting out. You see from this conversation that his reasoning power is not impaired, but that he is a person whose mind is dominated by a definite idea, and that is the characteristic of paranoia. Paranoia is a mental state that is dominated constantly by a fixed delusion, which may have some origin in fact or may not. Here it is

a delusion based on an hallucination of hearing. If it stood in some special relation to his general run of thought, it would not necessarily come out in conversation, and we might have to search for it; but here is a delusion that is always present, and he talks about it without the slightest hesitation. He comes in here and talks to us; while you or I, if we had the slightest suspicion that any person had a grudge against us, would not speak of it to a large audience as he does. So this idea so completely dominates his mind that it is the only thing that he thinks of.

CASE II.—This case is one that I think we shall have difficulty in discussing without exciting our risibility. The patient believes that she has inside of her a nest of frogs and that she is passing frogs, but has not succeeded in passing the original one as yet. I will ask you to control your natural tendency to laugh, because should you laugh at her we will at once lose any influence we may have over her. I will tell you about the treatment as she sits here. A case of this kind must be influenced through the mind, for her trouble is of a mental character. You will see the importance of treating a patient with delusion through the mind.

This patient states that for the past two years she has been troubled in her stomach and with her digestion. Seven years ago she passed the change of life. She did not suffer much at that time. She has always been healthy. She has had twelve children. She thinks that the origin of her trouble lay in swallowing a frog. She did not see it, but she felt the slimy thing going down her throat, although it did not scratch her at all. She did not at first think that it was a frog; the idea that it was a frog came to her gradually, and it was only at the end of three months that she was compelled to go to a doctor on account of what she felt inside,—that was heavings, pain, and a stinging feeling all through. Her digestion was not out of order. She got very sick by the end of three months, and went to several doctors. One of these helped her a good deal. He told her she had passed the frog, and for a time persuaded her and relieved the symptoms, but now she thinks it is there yet because she can feel it. She thinks there are several of them; she hears almost constantly the croaking of frogs. This croaking seems to come from her stomach and from the walls around her. She has also the feeling of everything about her being covered with dust. Her clothes are dusty, the bed is dusty, and she always sees a cloud of dust like a fog in the room. This she sees more at home than when she is out. She says that there is a feeling of heaviness in her feet, and when she comes to take off her shoes she finds them dusty or

sandy ; her hands are similarly affected ; in fact, everything about her seems dusty. In addition to this, there is a disagreeable odor which she thinks comes from the frog. Her water is especially offensive. She has peculiar sensations down her side and back.

You hear, gentlemen, that to her these symptoms are very real. The doctors she has been to have merely laughed at her,—all except the one who did her so much good. These symptoms are all symptoms that affect the various sensory organs. Smell, sight, hearing, and touch are all affected. They are of the nature of subjective sensations without any objective cause. They are hallucinations. Her explanation of them is a logical but a mistaken one, and is, therefore, a delusion. Such symptoms referable to the body and explained by the patient in one way or another should always lead us to investigate the condition of the nervous system. The first thing we would think of would be chronic multiple neuritis of alcoholic origin, with hallucinations and delusions ; but she has not been in the habit of drinking, for liquor does not agree with her ; so we cannot assign any peripheral condition as a cause. There is no organic condition of the spinal cord which could cause such symptoms. We must next think of the parts of the body affected at the beginning. The origin has evidently been in the stomach, and that is the part we propose to attack. There we will make our applications to destroy the animals which worry her, and we will be sure to find that she passes something in the nature of an animal, as she supposes. She has been directed to examine her stools. I will direct her to continue watching, and if she passes anything of the nature of a frog, she will bring it to us. In the mean time, on the theory that her belief is a true one, we are going to attempt to kill the frog that is in her stomach. You know there are cases of pregnancy occurring outside and not in the uterus ; and you know that under those circumstances it is possible for the child to come to life ; and when it does, the danger for the mother begins and may cause her death. So it is justifiable to kill the child in extra-uterine pregnancy. There is one good way of doing this,—namely, by galvanism. Now, we propose to apply the same method of treatment in this woman's case. We are going to apply a galvanic current over her stomach, which will not hurt her, but will kill the animals living in her stomach. Then by purgatives we hope to get rid of the débris. At the same time I will put her on general tonic treatment to get her health in better condition. After having got rid of these animals, we will get rid of the disagreeable sensation she has in her body and hands. [The woman leaves the room.] You may think that this is far-drawn, but you know

very well that if this woman is to be affected, it will be by none other than psychical means. To counteract this woman's delusion, it is by persuasion you must do it. Part of the treatment has been in bringing her before you and talking before her in this way. We may be able to counteract the delusion, and, if it is of an hysterical nature, to get the effect of hypnotic suggestion. I do not anticipate success, but I wish to give this woman some hope and an expectation of help. Here is a woman who, after the menopause in the course of two years, has developed a fixed and systematized delusion of a hypochondriacal nature. It is an interesting case, and illustrates the gradual development of a delusion in a person beyond the change of life, somewhat affected and discouraged by the conditions that surround her, and exhausted by frequent childbirth; a woman that in all probability has some predisposing influence towards mental trouble. It is undoubtedly a condition of beginning paranoia. This is the first delusion she has had, and it will undoubtedly grow. The delusion is based on an hallucination. The croaking she hears does not amount to a voice, but it may increase in intensity and change in character. She moved from one house because she heard the croaking so constantly around her coming from the walls and from her stomach. That is the way these delusions grow. You will see cases of paranoia with delusion thoroughly systematized; here it is only just developing. These delusions increase and become more fixed until the close of life. The insanity is not of a character to lead to physical disease or to death.

CASE III.—This woman has been sent here by Dr. Dudley, with the request that we look her over and make a diagnosis. I have not yet questioned her, and see her for the first time at present.

Q. How long have you been sick? A. About eight years.

Q. What is it that you first noticed? A. My head and heart.

Q. You have had headaches, have you? A. Yes, sir.

Q. Headaches and pains down your back? A. Yes, sir.

Q. You have had those constantly for eight years? A. For the last three, particularly.

Q. Is your pain so severe as to keep you awake at night? A. Most of the time I wake up about two.

Q. You mean you are awakened by the pain? A. I don't know whether by the pain or whether some one wakens me; I suffer after I awake.

Q. How do you suffer? A. I do not know what it is; my heart beats and I feel sick all over.

Q. You think you are awakened by some one? A. Yes, sir.

Q. Do you think it is any one in particular who is present? A. I do not know that it is,—it is a mystery.

Q. You think that this individual has been acting upon you for some time,—two or three years, or longer? A. About eight years. I do not know whether it is the same person or a number.

Q. You think it may be a number of persons, do you? A. Yes, sir.

Q. Have you had good health for eight years? A. I have been sick in bed for three years before Christmas.

Q. Had that anything to do with it? A. Yes, sir; that influence kept me in bed, and it has caused the impediment in my speech and made my teeth ache.

Q. You seem to talk clearly. A. No, I do not talk clearly; and now while I am speaking I am in a nervous condition.

Q. That might be from the persons present? A. No, it is not in that way.

Q. Then you feel it is the influence that makes you nervous? A. Yes, sir.

Q. How does this nervous influence act upon you? A. It seems to act everywhere I go. I have been to see a lady friend, and she said perhaps I brought my influence to her.

Q. Was she aware that you had this nervousness? A. Yes, sir; she knew of the shock of electricity I got.

Q. Then it is electricity, is it? A. Most certainly.

Q. Did you ever see an electric battery? A. Yes, sir, about seven years ago; a gentleman tried it on me, but it did not affect me.

Q. Did you think that shock seven or eight years ago started this? A. Oh, no.

Q. In regard to this influence that has been acting for eight years, have you always considered it was doing you harm? A. Yes, sir; it is either jealousy or envy.

Q. Is this the only interpretation you can give it? A. Yes, sir; I feel as if some one was drawing my life away from me. I feel nervous now, and my heart beats. Last night I went to see a friend, and the only way I could stop it was by pushing the drum of my ear.

Q. Have you ever taken anything to avoid the electricity acting upon you? A. Yes, sir; I have tried everything. I used to let cold water run on the back of my neck.

Q. Do you think that relieved you? A. It did for a little while. I drank a good deal of cold water and used all that.

Q. Have you ever thought of wearing a rubber band around your

neck to insulate your body? A. No; because this has worked me so that I have not a cent to my name.

Q. Now, you speak of this thing having possibly been brought on by envy and jealousy; do you know any one who is particularly jealous of you? A. I do not know any gentleman at all; if he is, he has not shown himself.

Q. What are they envious of? A. Well, my father married three times; and, perhaps, it might be from that cause. I do not like to commit anybody.

Q. Is your step-mother disagreeable? A. Yes, she is; but there is no occasion for any one to be disagreeable with me.

Q. Your stepmother is still living, is she? A. Yes, sir.

Q. Have you thought that perhaps she might have something to do with this? A. Yes; when I found it out I went to Captain Murray, of the Thirty-sixth Precinct police station, and told him at that time if this thing was not stopped I would be in the insane asylum; but I do not think anything could make me insane, because my brain is as clear as amber. There is a doctor living next door, and it may have come from that.

Q. Had you complained to the captain of the police court before that time? A. Yes, sir; I went down to Captain Murray. He laughed, and sent one of his officers with me to the car and sent me home. They all laugh at me, my daughter and all of them.

Q. Did it ever occur to you that the reason they laughed at you was not because they disbelieved in your symptoms and feelings, but because the explanation you give is the wrong one? A. No, because it is true; and I know most people do not care about the truth. They had that influence over me last night. I did not feel angry. When I came in the hall door I rang the door-bell, because it was troublesome to take the key out, and so I went in the hall door; and the woman came to the door and asked me why I did not use the latch-key. I said I did not use it because I did not feel like it.

Q. That might have happened to anybody. What influence can you ascribe to that? A. When I came down-stairs that severe pain came over me.

Q. Have you had any nervous symptoms at any time that disturbed you through the influence of these people? A. I know that any change that comes into my head is through the phonograph. When I sit down they commence talking to me. It annoys me very much.

Q. What do they say? A. Nothing in particular, always the same thing over and over again.

Q. Are they disagreeable or unpleasant things? A. Mostly unpleasant; therefore, it has disturbed me a great deal, and makes me very nervous.

Q. Have you noticed that people in the street look at you as they go by? A. Yes, but I do not mind that at all.

Q. Do you think people who are strangers pick you out? A. I do not know.

Q. You never heard people talking about you in the street-cars? A. I do not care about what people say, but the influence I am under I do not like, and those pains and aches I do not like. [Patient laughs.]

Q. That strikes you as amusing does it? A. No, something else strikes me as funny.

Q. You have things funny come into your mind, do you? A. Yes, sir. [Patient leaves the room.]

Gentlemen, this is a very clear example of the course of a delusion of persecution. I was not aware of the character of this patient at all when I brought her in. You see how the delusion is carried into every line of thought. You see the influence of the mind constantly dwelling on one thought. Nothing happens that she does not explain in a way related to her delusion. She comes in at night, rings the front door-bell, the servant remonstrates, and she flies into a rage. Instead of regarding that as a natural occurrence, it must be explained by the influence making her angry, and it is the same influence which caused the pain in her head, which followed the anger. You see how beautifully this case illustrates the fact that a delusion may become fixed in the mind, may be systematized, and so brought into relation with every effort of life that nothing can occur without reference to it. She believes there is a phonograph in her head, and every thought is referred by her to that phonograph; and you see how, after a time, hallucinations come in and support delusions. You see how she has acted on this delusion. She has gone to the police and asked protection, and has done what any of us would do if we were absolutely certain that particular individuals were exerting a malign influence on us. She believes that this persecution must be due to jealousy or hatred. Of course, one person is not jealous or envious of another without cause. You have noticed that she alluded to herself as particularly fine looking, and remember she said young men had noticed her on the streets. I do not believe she would have said that unless she considered herself a distinguished-looking individual. Thus a delusion of persecution often leads up to a delusion of grandeur,—to the idea that the person is a great, or rich, or learned person, a king, or even God.

Of course, you know perfectly well that the suggestion that I made of putting something on to insulate her from electricity was simply offered as a means of meeting a delusion by another delusion ; but this apparently ridiculous suggestion might possibly have some little beneficial effect upon her.

This is undoubtedly a case of paranoia. With regard to its causation, we see how little one can find. We are unable to get at the cause of the trouble. The only clue is the fact that the woman, who is forty years old, has had eight children. This was, of course, a great strain upon her, as all pregnancies are ; and it is not at all infrequent that these conditions develop in connection with marked anæmia and nervous exhaustion, and after repeated pregnancies. I think you could see that she is extremely anæmic. She is certainly very pale, and we know that anæmia is associated with palpitation of the heart, with headaches, and causes disturbances in the function of the spinal cord and the brain ; a feeling of weakness, a loss of self-control, and more or less of the symptoms which she complains of.

There is no cure for these cases, and the question comes up as to what we should recommend. These people are not dangerous in the community while their suspicions are not concentrated ; and, as this woman's suspicions are vague, I would not advise asylum cure. If, however, this individual begins to concentrate her suspicions upon some particular class and become, as she doubtless may, very much annoyed at their continued persecution, then at that moment she begins to become a dangerous person ; and then you must provide a place where she can do no harm. That is the point where we must make the distinction. You heard her say that she had made complaints to the police. These cases are so common that the Germans have given them a name (*Querulanten Irrsein*), complaining lunatics, and they are well known in all police courts. They are recognized by judges as insane, because of the character of their complaints.

THE TREATMENT OF MANIACAL DELIRIUM AND OF CHOREA BY CHLOROFORM AND MORPHIA, WITH REMARKS ON THE CAUSATION OF DE- LIRIUM TREMENS AND SEVERE CHOREA.

CLINICAL LECTURE DELIVERED AT THE LEEDS GENERAL INFIRMARY, ENGLAND.

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GENTLEMEN,—In the *Lancet* of April 15, 1893, I briefly described three cases of maniacal delirium which had been very readily cured by the administration of chloroform for a few minutes, preceded or followed by subcutaneous injection of morphine. There is, of course, nothing new in this combined method; but, so far as I can learn, it does not seem to be very commonly employed for that most troublesome and even dangerous disorder, violent delirium occurring in the course of acute diseases, such as rheumatic fever, chorea, pneumonia, and for the wild form of delirium tremens. I have known cases in which chloroform has been inhaled for hours together without any permanent effect; also, cases in which extraordinary doses of morphine have been required to compel sleep, and even with such doses some delay has occurred. With the combination of chloroform and morphine, all trouble and excitement are over—at least for a time—in a minute or two, as such patients seem to be “got under” with remarkable ease, and in the cases I have personally seen much more quickly than ordinary non-delirious patients. Chloral and bromides are much used for active delirium, but they often fail and always take time. Hyoscine is often used, but it is apparently more dangerous than morphine, and its dangers are, perhaps, not so well defined as those of morphine. We know that morphine is risky in cases of late granular kidney; of general nervous degeneration; in persons with idiosyncrasy; in some cases of bronchitis; but we hardly know in what cases hyoscine is dangerous. It was thought to be less dangerous than hyoscyamine,

which it has replaced ; but I have been informed of two deaths following its hypodermic use within an hour or two hours from the time of injection. Death seemed to be caused by paralysis of respiration. Hyoscyamine very nearly caused death in a similar way in the case of a gentleman aged about sixty, whom I saw some years ago. On the first day the delirium,—shouting, using coarse and abusive language, walking wildly about,—which seemed to be due to gout-poison, was relieved by the drug, several hours' sleep being obtained ; but on the second day, the injection of the same dose ($\frac{1}{80}$ grain) was followed, indeed, by sleep in a few minutes, but with absolute cessation of respiration, the pulse continuing full and quite unaffected. Smart shaking roused both brain and medulla ; inhalation of ammonia (common washing liquor) and injection of strychnine restored the breathing in half an hour ; he then slept calmly for two or three hours, but was no better on awaking. I have, however, seen exactly the same effect produced by chloral in a lady aged seventy, whose respiratory centres were damaged by pneumonia which had lasted a week and had followed a rather severe fall on the back. Fifteen grains of chloral and fifteen grains of potassium bromide were given together to induce sleep ; when the brain slept, the respiratory centre slept with it and was aroused with difficulty. Possibly morphine might have had a like effect. I have not yet had occasion to try the compatibility of hyoscine with chloroform, but the resident medical officer of the infirmary (Dr. F. A. Roberts) has recently injected $\frac{1}{100}$ grain of hyoscine after chloroform inhalation, to control maniacal delirium occurring in a patient, aged thirty, with disseminated sclerosis. The patient awoke when the chloroform was discontinued, but soon slept and remained asleep two hours. The following case, notes of which have been kindly given to me by Mr. W. H. Brown's house surgeon, Mr. H. T. Maw, illustrates the superiority in certain cases of morphine to hyoscine. Richard H., aged sixty-three, was admitted on June 28, 1893, for fractured neck of femur. Next day he was delirious. Hyoscine, $\frac{1}{80}$ grain, was injected at 10 P.M., and again at midnight ; on the 30th at 11 A.M. ; $\frac{1}{100}$ grain was injected at 11 P.M. ; on July 1, at 9 A.M., 4 P.M., and 8 P.M. ; on the 2d, at 9 A.M., all with very little effect. At 9.30 P.M., the patient was put under chloroform and $\frac{1}{2}$ grain of morphine was injected ; he slept nine and a half hours. On the following night morphine was again injected without chloroform. After this he recovered without further treatment.

Dr. Finlay has kindly informed me that he saw a case treated in the Middlesex Hospital twenty years ago by the combination of chloro-

form and morphine, and there can be little doubt that many practitioners must have been compelled to use it at times by sheer necessity, as, in fact, was the case with myself in the first instance. A tradesman, aged thirty-two, was being attended for pneumonia by Mr. C. V. Newstead, of Leeds. On the third night the patient became delirious, shouting "murder" at the top of his voice and very rapidly, in such sort as to rouse the entire neighborhood and cause considerable excitement. He utterly refused a proffered draught; and when, being held down by force, one-fourth of a grain of morphine was injected into his arm, he interpreted the act as additional reason for alarm, and shouted even more loudly and quickly than before. Naturally, therefore, we turned to chloroform to obtain at least a short respite, and when the chloroform was stopped, after a few minutes, to our great satisfaction he continued to sleep, and slept for three hours. He was then no less noisy. The chloroform and morphine were repeated; he slept two hours. Mr. Newstead then repeated the chloroform and injected half a grain of morphine; after this dose he slept for three hours, and when he awoke the pupils, which were at first very distinctly dilated, had become small; he was quite sensible and free from delusions and delirium and remained free, recovering in the ordinary way.

The next case in which the combination was tried was that of a man aged twenty-one, who, beginning with acute rheumatism, quickly developed, first, chorea and then mania. This patient also shouted "murder," and fought and struggled greatly. In three or four minutes after chloroform and morphine, he was fast asleep, and slept for several hours. Chloral and hyoscine, which were separately tried after this first sleep, entirely failed; but under repetition of the chloroform and morphine treatment, his case gave no further trouble and no special male attendant was required. .

The case mentioned by Dr. Finlay was "a lad of sixteen, who came into his hospital with severe chorea in which he became maniacal. He had chloroform inhalation and subcutaneous injection of morphine thirty-two times, went through pericarditis and pneumonia, and made a good recovery."

Nothing can, in fact, be more satisfactory for severe chorea; chloral and bromide in large doses twice hourly are next in order of service; hyoscine (without chloroform) and conium I have long given up; in arsenic I have no confidence except as a tonic in chronic cases; to me arsenic has, in fact, seemed much more than useless in bad cases. The following recent instance illustrates the value of the combined treatment and its power of arresting not only delirium, but also the exces-

sive movements which are so painful to witness and so hard to control in bad chorea. Greatly preferable to mechanical restraint or even to the most careful and gentle nursing, and, as it seems, more rapidly curative than any other method, the treatment might well, perhaps, be resorted to in cases of even much less intensity than the one to be related. The share of diet and of prolonged rest of muscles and nerves in the cure will be considered presently.

Sarah C., aged seventeen, was admitted on November 28, 1893, with slight pain in the right wrist and in the knees and chorea of moderate degree. There was an apex systolic bruit; urine albuminous; specific gravity 1020. Temperature 99° F., on the evening of this day, but afterwards steady at 98.4°–98.5° F., both morning and evening. Pulse about 100; respiration, 24 to 28. When seven years old she had acute rheumatism, and had frequently suffered from pains in joints. Two years ago she was in bed for six weeks with chorea. She had had no fright or shock, but had often got wet through during her walk of thirty minutes to and from work. The chorea came on gradually a fortnight ago, after pain in her limbs had existed for a week. She had taken ordinary diet before admission,—meat, bread, butter, etc.

Drug Treatment.—Sodium salicylate, 10 grains, every four hours; chloral hydrate, 10 grains; potassium bromide, 15 grains, if required, every four hours.

November 30.—The movements are not lessened. At midnight morphine, $\frac{1}{4}$ grain, was given subcutaneously. She slept afterwards at intervals.

December 1.—Patient had a very restless night. Chloral, 20 grains. Slept about two hours during the night.

December 3, 1 A.M.—Chloral, 20 grains. Slept from 2 to 7 A.M.

December 4.—Very restless and noisy. Chloroform inhaled, and chloral, 10 grains, was given subcutaneously, but with no effect. 3 A.M., chloroform inhalation, and morphine, $\frac{1}{4}$ grain, subcutaneously. She slept three hours, but not continuously. 10 P.M., chloroform inhaled; morphine, $\frac{1}{4}$ grain, subcutaneously. Continuous sleep for three hours followed.

December 5, 1 A.M.—Not improved. Movements of head, trunk, and limbs as violent as ever. Chloroform with morphine, $\frac{1}{4}$ grain, subcutaneously, procured only half an hour's sleep. 4.15 A.M., chloral, 20 grains, injected into thigh during chloroform inhalation; patient slept from 4.45 to 8 A.M. On awaking she was no better. The resident medical officer (Dr. F. W. Roberts) had proceeded cautiously with morphine up to this point, but it became evident that larger doses were

required, as she rambled very much in her talk and replies, and the rapidity and range of the movements made it difficult to keep the patient from being thrown out of bed every minute or two; moreover, she was becoming bruised and excoriated. I, therefore, ordered one-half a grain of morphine to be injected, and sleep to be then initiated by chloroform. This was done at 10 A.M. on December 5. She then slept for nine hours (not quite unbrokenly) and awoke decidedly improved. The after-progress of this case to its recovery is, perhaps, worthy of remark from another point of view. It has for a long time been to me a cause of wonder that many children who have been admitted to the hospital with chorea of very moderate intensity have got worse instead of improving, and this no matter what the drug treatment may have been, and also in the absence of drug treatment. In fact, drugs seemed to have little or no influence,—potassium bicarbonate, or, even if there was a slight rise of temperature, salicylate of sodium, iron, arsenic in small or (in a few cases only) in large doses,—none seemed to make any difference. In the case of Sarah C., the extraordinary steadiness of the temperature in the worst as in the lightest phases of the motor disorder—the morning and evening chart-marks being often exactly alike—arrested my attention. Remembering the disturbing influence of some brain disorders upon temperature, it occurred to me that very possibly the temperature which would otherwise exist in rheumatic chorea was inhibited by the irritated brain itself, and that the poison causing acute chorea, though producing some of its other results, might fail to produce the temperature. Whether this idea is right or wrong, the treatment based upon it succeeded in arresting all the movements in this case very quickly. It will be seen from the appended table of temperatures, etc., that on December 11 plain diet,—that is, bread and butter, rice, tea, and broth,—with an egg daily, was begun. On the next day the movements were worse, and the report of the night-nurse runs thus: “Wandering during the night; imagined she saw various things, and said we were all laughing at her, when the ward was perfectly quiet.” Strict milk diet was at once resumed, but no drugs were given. A week afterwards bread was added to the milk; not until December 30 was dry bread allowed. Butter with bread seems very distinctly to restore the pain and swelling in *some* rheumatic persons. I have tried many experiments upon this point, temporarily complying with the urgent request of patients; hence its exclusion in this (rheumatic) case. It must be said, however, that the patient during the severe part of her disease took milk only, being, in fact, unable to take anything solid. It is very probable, therefore,

that another cause of the increase of the bad symptoms of violent chorea is the chorea itself. There is considerable disintegration of muscle, but chemical mutations in the liver and elsewhere are not now perfect; other rheumatic affections—arthritis, carditis—very often relapse if movements are made too soon. A prolonged rest is curative, but it must be perfect physiological rest; not mere mechanical restraint of the peripheral evidence of central over-activity. Under such restraint the excessive muscular action is not lessened, the energy is merely changed into tension of cords and bandages; the formation of poisonous products is unchecked.

CHARTS IN THE CASE OF SARAH C.

Date.	Temperature.		Pulse.		Respiration.		
	M.	E.	M.	E.	M.	E.	
Nov. 28	99.2	...	92	...	28	
" 29 . . .	98.6	99	...	96	...	28	Sod. salicyl., gr. 10, 4 h.
" 30 . . .	99	99	100	84	...	24	Sod. salicyl., gr. 20, 4 h.
Dec. 1 . . .	99	98.5	102	106	24	28	Milk only.
" 2 . . .	98.5	98.5	96	98	24	...	Bread and milk.
" 3 . . .	97.8	98.4	88	100	22	24	Tea and rice.
" 4 . . .	98.4	98.4	98	102	...	24	Omit salicylate.
" 5	98.4	...	108	...	24	
" 6 . . .	98.4	98.4	100	100	...	20	
" 7 . . .	98	98.8	100	88	...	20	
" 8 . . .	98	98	80	80	...	20	
" 9 . . .	98.4	98.2	84	84	...	24	
" 10 . . .	97.4	97.8	88	88	
" 11 . . .	98.4	98	84	84	M. ferri perchl. t. d. Plain diet—eggs.
" 12 . . .	98.4	98.4	Omit. Milk only.
" 13 . . .	98.4	98	76	72	28	24	No medicine.
" 14 . . .	98	98.4	72	72	
" 15 . . .	98.2	98	80	72	
" 16 . . .	97.4	98	72	64	
" 17 . . .	97.2	97.2	76	72	
" 18 . . .	98	98.4	84	72	20	24	
" 19 . . .	97.4	98	60	72	Ferri tartrate, gr. 5; potass. bicarb., gr.
" 20 . . .	98.2	98.4	60	78	20, t. d. Bread
" 21 . . .	98	98.6	72	50	and milk.
" 22 . . .	98	99	72	78	24	24	Tea, bread; no but-
" 30 . . .	97.6	97.6	...	80	...	28	ter.

Chloral, morphine, aperients, etc., are omitted from this list.

At the same time there was in the hospital a child who had been choreic more or less for a whole year. Mary E. H., aged five, was admitted on October 26, 1893, with a moderate chorea affecting face, trunk, and limbs. She was fairly bright and intelligent, of quiet manner, and did not look ill. Temperature, 97°–98.4° F.; pulse, 80; respiration, 20 to 28 per minute. As she had been rickety and never

strong, she was treated by syrup of phosphate of iron, cod-liver oil, and full diet. Under this treatment she got distinctly worse, and the movements were more frequent and of wider range. The temperature, at first varying from 97° to 99° F., became steadier at (about) 98.4° F.; pulse varied from 64 to 84; respiration from 20 to 28; an old mitral bruit was unaltered. After six weeks of treatment she went to a Convalescent Home on December 2, but was sent back in a few days, as the movements were still worse; she was restless at night, and began to have a difficulty in taking food with her own hands. The temperature had been lower—97°–98° F.; occasionally, 96° F. On December 12 arsenic had been ordered at the Home, but as she was gradually getting worse day by day, on the 18th she was put upon strict milk diet and potassium bicarbonate five grains in camphor water thrice daily. Improvement at once began. On December 25 the excitement, and perhaps the diet of the children's ward, produced an evening temperature of 102.4° F.; pulse, 112; respiration, 28. Next day, however, the temperature was 98° F., morning; 97.4° F., evening; pulse, 100; respiration, 24. Bread-and-milk diet ordered, with one egg daily. On December 30 the movements had entirely ceased; she could walk quite naturally, and use her hands for every ordinary purpose.

It is, of course, perfectly true and undeniable that many cases of chorea recover upon ordinary diet, just as many cases of typhoid fever and of rheumatic fever recover eventually upon a diet very far removed from the severe simplicity of the milk diet, though probably few will assert that the mixed and free diet is the most comfort-yielding for the attendants and doctors in the great majority of such cases; and it is possible that many patients, and perhaps even whole classes or races, may be unable to digest milk, boiled or unboiled, hot or cold, flavored with brandy or not, with aerated water or without it; there are exceptions to almost everything one can state, and my present contention relates only to observations upon patients in a Yorkshire hospital, including however, persons of very different personal characteristics—Northern and Southern English, Irish, Scotch, Jewish, etc.

Delirium becoming intense at the crisis of pneumonia readily yielded to the chloroform and morphine treatment. Albert F., aged thirty-three, an iron-worker, was admitted on January 8, 1893, for acute pneumonia. He walked into the hospital without difficulty. He was a resolute or obstinate man, drank much beer, had had a cough for some months, was off work three weeks ago with a severe cold, then had a rigor, and was ill for a week, but not in bed. On January 5 had rigor

again and was unfit for work that day ; went to work on the 6th, but was behaving in a delirious way in the street that night. Tubular breathing, etc., in right upper lobe. Urine contained a little albumen and indican. He showed the almost offensively assertive disposition which sometimes precedes outbreaks of delirium in pneumonic patients (one man, in whom this brusque and abrupt mode of denying that he was weak, or ill, or fit to be kept in bed was observed, leaped out of the window half an hour afterwards), denied that he was weak, and, as the notes say, rather overdid the appearance of energy. During the night he was delirious, getting out of bed, wanting to go to work, etc. On the next day (9th) the temperature fell rapidly to 99° F., the pulse from 112 to 88, respiration from 50 to 32, but the delirium increased. At 8.30 P.M., as the bowels had not acted, a glycerin enema was ordered, but he became very violent and noisy when the attempt was made to administer it. The house physician (Mr. E. Baines) injected $\frac{1}{2}$ grain of morphine at 9 P.M. and again at 9.30, but when seen by me at 11 P.M. he was even more violent and required several persons to keep him in bed. He struggled furiously to avoid chloroform, but was overpowered and put under it ; $\frac{1}{2}$ grain of morphine was then injected, also 20 minims of brandy, as his pulse was found to be rapid and variable in force ; the bladder, which, though distended nearly to the umbilicus, he had refused to empty by micturition, was emptied by a catheter ; the rectum was washed out ; the chloroform was then omitted ; he slept until 3 A.M. He was then again violent and noisy ; the morphine ($\frac{1}{2}$ grain) *plus* chloroform treatment was repeated ; a nutrient enema of egg and milk was given ; he slept until 7.30 A.M. At 8 A.M. he was still very much excited, but took some bread and milk, went to sleep soon afterwards, and awoke in an hour quite sensible and calm. He slept much during the rest of the day, but awoke occasionally and took food. The pupils were contracted all day. He left the hospital in a fortnight.

In the next case delirium tremens came on in a man a few days after drinking heavily. Smaller doses of morphine sufficed in this case ; there was some albuminuria.

William C., aged thirty-five, was admitted November 18, 1893. He was a man of some ability, dark hair, large head and face ; of American aspect. He had been drinking freely champagne, rum, etc., and at last had swallowed a compound of petroleum and corrosive sublimate. An emetic was promptly taken and the stomach was subsequently washed out ; apparently very little of the poison was absorbed ; the gums were never affected. On the next day he was rather tremu-

lous; the temperature, which was 95° F. on the night of admission, rose gradually during the day to 99° F.; pulse, 120; he was content with milk and soda-water. This was continued on the 20th. Temperature fell to 97° F.; pulse, 84. On the 21st, tea, bread, rice, and broth were allowed; his wife (as was afterwards discovered) also brought in a pork-pie, of which he ate a small piece. At 3.30 A.M. on the 22d he awoke and wished to leave the hospital, on the plea that if he was not at home at 6 A.M. the bailiffs would be put in. At mid-day his friends came and assured him all was right at home, and he seemed satisfied. He ate rice for dinner at noon, and bread and butter with tea at 4 P.M. At 5 P.M. he leaped out of bed, saying it was full of pikes; he shouted and became hard to restrain. At 6.50 P.M. hyoscine, $\frac{1}{100}$ grain, was injected subcutaneously, but it produced no effect for nearly two hours; he then slept uneasily, moving about and moaning at times. Soon after midnight he was again noisy and maniacal. He was put under chloroform; $\frac{1}{4}$ grain of morphine was injected subcutaneously; the sleep continued less than two hours. At 7 A.M. (23d) $\frac{1}{4}$ grain of morphine was injected, the strict milk and aerated water diet enforced, and an effervescing saline mixture was given every four hours. His tongue was dry and furred; he complained of bad tastes in his mouth, and of sour fluid rising into his throat. Pulse quiet, regular, of good volume. He was removed to a private ward, with a male attendant. The delirium, however, did not return; the attendant had nothing to do. 24th. Urine, specific gravity 1010; much albumen; also indican and ptomaines or peptones. 25th. Had vomited. Urine, specific gravity 1006, albuminous. Ordered mustard-leaf to epigastrium; zymised milk. 27th. Urine much less albuminous; specific gravity 1010. To have bread and milk, and next day rice and bread with butter. On and after the 25th the bowels had acted four or five times daily without aperients. Pulse about 65. 29th. Left the hospital, it was supposed in order to obtain alcohol, without which he said he could not be contented. There was still some albumen in the urine, but very little indican. With respect to the causation of the delirium in this case also, I believe that it resulted from a poison derived from undigested and fermenting food in the intestine. A large quantity of indican was found in the urine after the bread and pork-pie were taken, though it had not been present in the urine previously. The cells of his digestive organs had not recovered from the alcohol and the poison; the food was, therefore, not acted upon by his natural ferments, but by ferments casually swallowed with the food; hence the foreign products, which, in turn, imperfectly separated or destroyed the temporarily inefficient liver,

poisoned the blood and brain; they probably caused the albuminuria also. And there seems to be reason for suspecting that the delirium tremens which comes on after an accident in an alcoholic patient is due not to the deprivation of alcohol (William C. got well, as many other patients have done, without any), but to the comparative over-feeding or inapt feeding which such patients often receive. I have seen a case of enteric fever, and in this disease there is usually impaired digestive power, in which delirium continued for several weeks after the temperature was normal, under the influence apparently of a liberal diet,—meat, vegetables, etc. Theoretically, one would expect disturbance somewhere; the weakest part might be expected to suffer most; the result would not be the same in every case. In other patients a mere relapse of the fever would have been caused; in some it may even be granted that *no* obvious symptoms of disorder would have been caused; but the experience of all of us is too strong to be overborne by occasional exceptions occurring in persons of extraordinary construction. In every disease due to micro-organisms there are at the very roughest estimate at least two variables,—(1) the original chemico-vital structure of (each organ of) the patient; and (2) the surrounding conditions, sometimes more favorable to the micro-organisms, sometimes to the patient.

If the delirium in the case of William C. had been due to the presence of a micro-organism such as the bacillus discovered in a case of acute delirium by Rasori, it seems improbable that the morphine could have cured the disorder, as it did, in a few hours. But if the delirium was due to products made in and absorbed from the gastro-intestinal tract, it is readily intelligible that morphine might act as an antidote to their influence on the brain until the supply of the poison was exhausted or their source expelled. If the poisons are neither expelled nor antagonized they may behave like chloroform, which causes, first, delirium and afterwards a coma, which continues as long as the supply of poison is continued. Ten or fifteen years ago consultations in cases of delirium occurring in typhoid fever were not uncommon, now they are rare, apparently in consequence of the more general adoption of a milk diet.

Inquiry must always, if possible, be made as to the capacity for morphine. When, as often happens, the patient has never taken morphine, the capacity of his parents or other members of the family, especially any whom he may resemble in personal characteristics, should be inquired about. In the case of a young man with typhoid fever, the following incident occurred in consequence of a want of this precau-

tion. As the patient suffered from insomnia and restlessness bordering on delirium, it was proposed to give him a hypodermic injection of morphine. His friends were asked collectively whether he had any peculiarity with respect to morphine, but they did not know, as he had never taken any. As the family was rather liable to nervous disorders, only a small dose, one-sixth of a grain, was injected. In less than an hour severe convulsions occurred. These gradually diminished in intensity, but movements continued in the left arm and leg for two or three hours. The pupils were sharply contracted. The mother then stated that she had always been made ill by morphine. The patient resembled her very closely in complexion and features, and doubtless in this peculiarity also.

Finally, it is probable that in most cases of chorea, delirium tremens impending after accidents, pneumonia, typhoid fever, etc., a strict dietary, adjusted to the diminished digestive capacity of the patient, will, by preventing the excessive formation of brain-disordering ptomaines, prevent the delirium. But if the damage has already been done,—if the poisons are in the blood and you cannot get them out, but must, on your part, introduce a counter-poison,—then the following rules, or some like them, may serve to guide you in these sometimes rather bewildering circumstances :

1. Make reasonably sure that there is nothing in the state of the kidneys or of the respiratory and the circulatory organs, including their nervous apparatus, contra-indicating morphine.

2. Put the patient under chloroform.

3. Inject $\frac{1}{2}$ grain of salt of morphine ; or, if the morphine capacity of the patient is known to be good, $\frac{1}{4}$ or $\frac{1}{2}$ grain into the arm.

4. Continue the chloroform if it is necessary to empty the bladder, or to wash out the rectum, or give a nutrient enema. If none of these are required, or when they have been done,—

5. Stop the chloroform ; keep the room and the house perfectly quiet.

6. If the patient wakes in a short time *without external cause* and is again violent, repeat the chloroform and a slightly larger dose of morphine. (If he has been awakened by noise and is not too violent, wait some minutes for the possibility of his sleeping again.)

7. If he now sleeps three or four hours the proper dose of morphine has been found. It may be repeated, with chloroform, as often as required.

8. But if the patient again awakes without external cause in an hour or two, is as violent as before, and has *not* contracted pupils, the

dose of morphine may be still further increased to half a grain. If the pupils had become decidedly contracted by the smaller dose, I should not push the morphine; but so far no such case has occurred to me. The dose that contracts the pupil calms the brain.

9. If the delirium is much less violent when the patient awakes, he may, if he will, take some warm milk with or without bread. This is sometimes followed by sleep without further drugs.

10. The treatment is dangerous if the kidneys are inefficient, so that little or none of the drug is eliminated by them; or, if cardiac and respiratory nerve-cells are degenerated; also, in persons with idiosyncrasy.

11. In doubtful cases both chloroform and morphine must be used very cautiously. In uræmic convulsions and in uræmic mania, morphine is probably wrong, unless the pupils are dilated by the toxins producing the disorder, then morphine may be right.

12. Only in very rare cases is anything in the nature of mechanical restraint justifiable in ordinary practice. It is to be looked upon as a signal and painful "confession of failure."

THE BONUZZI TREATMENT OF LOCOMOTOR ATAXIA; REMOTE EFFECTS OF INJURY TO THE LUMBAR SPINE.

CLINICAL LECTURE DELIVERED AT THE MEDICO-CHIRURGICAL COLLEGE HOSPITAL,
PHILADELPHIA.

BY SAMUEL WOLFE, M.D.,

Professor of Physiology and Clinical Professor of Nervous Diseases, Medico-Chirurgical College.

GENTLEMEN,—We do not expect anything like rapid results from the treatment of any disease of a neurotic character, yet whether it is due to the treatment or to the incidental changes which always go with chronic diseases, there is evidence of improvement in this case of locomotor ataxia which you saw me treat by a certain method. His movements are more regular than they were a week ago. He stands on one leg now better than he did then. You will also remember that it was last week that he was unable to get his foot from the floor, but now his coördination is better than then. Now, I think we are in some measure, at least, justified in attributing these changes to the treatment which we had put him on, and it is certainly encouraging to continue this treatment and see the ultimate result. You will also remember that this treatment has been introduced within the last few years by Bonuzzi, an Italian physician, as a substitute for the suspension treatment. The suspension treatment has been in practice for a long time both in Europe and in this country, and has been of benefit to some of these tabetics. I do not know that we have any records of positive cure, but when we can improve the coördination, and when we can, to a certain extent, rid these patients of their lightning pains of progressive character, we have accomplished pretty much all that lies within the range of our power,—within the range of therapeutics. This man has taken nothing at all internally. The only single drug that has done more good probably than any other is liquor potassii arsenitis. Other preparations of arsenic will do equally as well. This is a drug which is prescribed as a routine in these cases. It is not given in very large

doses, but persistently, four, five, six, or as high as fifteen minims, so as not to produce poisonous effects. It is not desirable to give it in as large doses as in chronic malaria. It is usually better to prescribe it in gradually increasing doses, as three or four minims for one month and four or five minims for the next month, and in this way keep it up for a year or more. It is seldom we give over fifteen drops at a dose. Under this drug patients do fairly well. The disease is frequently kept from making rapid progress, and the symptoms may even partly disappear. This patient's power of coördination is much better; his movements are better. We shall ask him to come back in one week.

He speaks of lightning pains. He has more marked sensations during the winter than during the summer, and during the past week, when the temperature was quite low, he has had considerable pain, referred especially to the left knee; and the pains have been especially persistent one day of the week, and that day was quite a cold one. This probably was due to the cold producing a form of pain closely akin to rheumatic pain. The pain usually extends through the whole leg. In this patient the pain is often right around the knee, sometimes in the ankle, sometimes in the foot, and sometimes in the toes. As a usual thing it does not extend through the whole leg at once with him. It is very likely that this pain is of the nature of lightning pains, slightly exaggerated in this case by rheumatism. This patient has had mild crises, which go with this disease, and in some cases are decidedly severe. These crises are certain symptoms which develop and have a certain connection with some of the internal organs, stomach, bowels, larynx, etc. Some of these patients are troubled very much with severe attacks resembling colic, which would be very difficult to diagnose from an ordinary attack of colic if the other symptoms were not present. There are laryngeal crises; attacks which are somewhat similar to croup or laryngismus stridulus. These patients are troubled with a great deal of dyspnoea, spasmodic cough, and pain around the larynx. There are also often genito-urinary symptoms. In a certain proportion of cases they never occur. They are, it may be, a discharge through the nerves of these organs, which gives rise to the spasmodic symptoms and pains; and these pains probably have a close relation to the lightning pains in the limbs which are supposed to be due to irritation of the fibres running through the sclerosed portions of the cord, in the course of the degenerative process.

CASE II.—This man, aged fifty-nine, was born in Ireland, and came to this country forty-five years ago. I have not seen him before this moment, and must extract his history in your presence. He is a

carpenter by occupation. About a year and a half ago he was lifting some heavy timber in a building, when a heavy joist fell across his back. He was not able to do anything for three months after the accident. Right after the accident he was unable to walk, but says he could freely move his legs while lying in bed. He then walked about with crutches. There are no evidences of a bruise or injury where the timber struck him. He had some pain in his back.

Now observe the condition of the spinous processes. In the dorsal region they are regular, lower down they begin to protrude. You will notice there is a decided protrusion of all the lumbar vertebræ; none in the sacral region. The lumbar vertebræ are displaced posteriorly and laterally. By rubbing down along the spinous processes we can see their lines better, owing to the red points produced over the successive spines. I think you will be able to tell, by looking closely, that there is a little lateral displacement. You will notice there is absolutely no tenderness to pressure. You will remember that this man was injured a year and a half ago, and that he was not able to walk for three months following on account of it. There is no pain on the application of hot water. If there is any obscure region of the spine hurt, you can detect it by running a sponge filled with hot water down the spine. The patient will complain of heat pain when he will not complain of pain from pressure. This is a delicate and important test to apply to these cases. At night he gets cramps in both legs, and all the time the anterior surface of both legs seems to be cold, even in warm weather. These cramps are most severe at night, and are confined to the thighs; they are not painful in character. He has had no trouble with his bladder. He was not knocked senseless by the blow, and at no time did he lose consciousness. These cramps have been getting worse and more or less constant since leaving the German Hospital, where he was taken immediately after the injury. There is no pain shooting down the back of the legs, there is no wasting of the muscles. The cramps and sense of coldness have been very constant. There is no sense of coldness to the touch, and there is no doubt that the symptoms are due to the injury which he sustained to the back.

The question that suggests itself is, How far are these symptoms due to injury of the spine, and how far to injury of the nerve-roots as they emerge from the spine? From the fact that this man was not absolutely paralyzed, we can conclude that there is no myelitis; and, as I find that there is injury to the bony parts in that region where there is no part of the spinal cord proper within the spinal canal, but where it is filled by a bundle of nerve-roots, I am inclined to the opinion that

the symptoms are due to an implication of these nerve-roots as they come from the spinal cavity rather than to an injury to the spine itself. It is a case in which the nerve-roots are involved in the inflammatory processes which have resulted from injury to the bones, ligaments, etc., in the neighborhood of the lumbar region of the spine. There is also some disturbance of sensation,—not to the extent of anæsthesia, but paræsthesia,—a disturbance rather than an abeyance of sensation. There are no bladder symptoms, no incontinence of urine, nor was there any immediately after injury, and no paralysis. I think we are warranted in coming to the conclusion that the symptoms are due to peripheral troubles,—to injuries outside of the spinal cord. The left knee-jerk is present, slightly increased. Even on the reinforcement, we cannot elicit any knee-jerk on the right side. There is no ankle-clonus on either side. The right side, remember, is the side on which we have no knee-jerk. On the left side, where the knee-jerk is present, we get no cremasteric reflex. Notice the entire absence of tone in this left dartos. Now let us test the cutaneous reflexes about the abdomen. Abdominal reflexes on either side are not at all well marked, also the epigastric reflexes are not well marked. The plantar reflexes are abolished. The only skin reflex present is the cremasteric on the right side, and that is somewhat exaggerated. In the irregular way in which the reflexes in this case are affected, I think we have some confirmation of the view that the nerve-roots are affected rather than the spine itself. If the spine were injured in the region of the lumbar enlargement, the reflexes would be exaggerated if that part of the spine were merely irritated; they would be abolished if it were destroyed. If the injury is above the lumbar enlargement, we would be likely to have the reflexes exaggerated for this reason,—the knee reflexes are due, so far as they are reflexes, to the lumbar segments of the spinal cord, but there is a more or less inhibitory power controlling them which must come down through the dorsal region of the cord from the brain. Here we have the abolition of one of the reflexes and the exaggeration of another. That would come through the peripheral nerves rather than through the centres.

Now, what are the principal changes going on in those nerves which are affected? On standing with the feet close together there is a tendency to sway. He does not stand well on one leg. There is a certain amount of incoördination. This may arise from faulty conduction of sensations through the nerves, as through the tracts in the cord, as you know is the case in locomotor ataxia.

Now, what is the nature of the inflammatory processes which are

going on in the nerves? The ligaments which hold the spinal column together and the muscles upon the back of the spinal column and all the tissues of that neighborhood were no doubt more or less bruised, and the inflammation involves the lumbar region of the spinal column rather than the lumbar region of the spinal cord itself. In this connection you will do well to recall the fact that the spinal cord ends on a level with the lower border of the first lumbar vertebra, and that the remainder of the canal is occupied by the roots of the nerves which have left the cord higher up,—have left the cord, but not the canal. This inflamed area of the nerve-roots and the nerves issuing from the spinal cord were caught, and shared in the injury. Under such circumstances you would naturally look for some tenderness of the trunks which were pressed upon. No doubt there are still inflammatory and degenerative changes going on. There is not sufficient to entirely impede the motor impulses, so that this man has perfect motor power, but his sensations are somewhat involved. There is sufficient interference with the function of these nerves to bring about such symptoms as cramps and such disturbances as lead to false impressions. His thighs feel cold to him, but they are not cold to the touch : the sense of coldness is subjective, not objective.

Now, what can we do for this man? The main treatment in a case of this kind would be to try to remove as much as possible all the effused inflammatory products. I think we ought to address ourselves as much to the spine as to the legs. He should, of course, put warm clothing to the limbs, and to all parts that are involved and of which he complained. He should have hot and cold applications to the lower part of the spine; the use of iodide of potassium internally; tincture of iodine painted twice a day over the whole region of the spine; hot sponging to the back for about five minutes, immediately followed by the cold douche. Have the water as hot as he can bear it; let the cold fall in a stream from a height—say a foot or a foot and a half—for a minute. This should be done once every day, and the best time of the day is in the morning, about two hours after breakfast. The tincture of iodine is to be applied to the back. This will have a tendency to tone up the vessels and stimulate the absorbents, and thus to remove the inflammatory products.

The following prescription will be found useful in promoting absorption :

R Pot. iodi., \mathfrak{z}_{ss} ,
 Aq., q. s. ad \mathfrak{z} ii.—M.
 Sig.—Gtt. xx in milk, t. d.

By giving the iodide of potassium in milk, it will better agree with the stomach than when given with water simply.

The question of diagnosis which may come up in cases of this kind where there is peripheral disease is, I think, very well illustrated in this case. If we did not have a history of this case,—if he had not been able to tell us anything about the injury to his back,—it would have been a much more difficult question of diagnosis. Finding the displacement of the vertebral processes which we find in this case, it is more or less difficult to decide whether such an injury involved the cord itself or the nerves. I feel justified in saying that up to this time this man has no central nervous disease, though that may take place in time. These nerves have a tendency to degenerate upward along the anterior and posterior roots, ascending up through the cord; so that ultimately we may have in this case a certain amount of sclerosis. We may have established later in this case, unless we can remove the inflammatory products, a condition which is very much more similar to locomotor ataxia than the symptoms present at the present time.

HEMIPLEGIA IN CHILDREN.

CLINICAL LECTURE DELIVERED AT LONG ISLAND COLLEGE HOSPITAL.

BY JOHN C. SHAW, M.D.,

Clinical Professor of Diseases of the Mind and Nervous System in the Long Island College Hospital, etc.

GENTLEMEN,—We will consider to-day a form of hemiplegia to which children are subject. We may have other forms of paralysis in children, but they are not hemiplegic in distribution. We may have hemiplegia from cerebral tumor or abscess; hemiplegia in acute poliomyelitis anterior—very rare; hemiplegia in hysteria—not common in children; hemiplegia post-convulsive or post-epileptic—these last two are functional disorders and are not permanent; hemiplegia from gross brain diseases other than those mentioned above. It is to this last that I desire to call your attention.

You must not lose sight of the fact that hemiplegia is only a symptom, and although it generally indicates a disease of the brain, there are exceptions, as when we have hemiplegia—that is, paralysis of an arm and leg on the same side—from a disease of the spinal cord, as in acute poliomyelitis anterior, or from an injury of one-half of the spinal cord. These cases are, however, not common.

This young man is twenty years of age; he is fairly intelligent. All that we can learn of his previous history is from himself. When he was seven years old he had a convulsive seizure, but of which he can give no accurate account. Since that time he has been weak on his right side; he had convulsions frequently until he was ten years old. Since that time he has had no convulsion. He has been able to work in a woollen-mill up to four or five years ago, when he could no longer do his work owing to a trembling which began in his right hand.

He is now partly paralyzed, as you see, on the right side. On his holding the right arm out, you notice a fine tremor. This tremor ceases entirely, or almost so, as soon as he rests the hand on his knee; if he again holds the arm extended from the body the tremor begins. If I now ask him, without moving his arm from the extended position, to

take hold of my hand, you observe that the tremor ceases at once. The tendon reflex is normal. The muscles on the right side—the paralyzed side—are smaller than on the left, but not markedly so; there is no contracture or rigidity at the elbow, wrist, or shoulder. He has a paralysis of the right sixth nerve. There is no optic neuritis or atrophy.

In this girl (second case), who is in appearance only a child, but is fully seventeen or eighteen years of age, I can give you no previous history. She is an inmate of the Kings County Hospital. I will ask you to contrast her with the other case. We find a right hemiplegia with a certain amount of contracture; you see the flexing at the elbow and the wrist. If you attempt extension of these parts you find the muscles very rigid, especially so at the wrist. It is impossible to overcome this contracture except to a very slight degree. There is only a slight difference between the muscular development of the two sides. Her intelligence is very limited; this is in marked contrast with that of the young man. She is subject to epileptic attacks. There is no trembling of any kind on the paralyzed side. (Fig. 1.)

Allow me to call your attention to another case by means of a photograph. The patient is now dead, and later I will show you the brain. He was an inmate of the Kings County Hospital; his age I cannot give you, but you observe that he was a young man, probably between twenty-three and twenty-five years old, when this photograph was taken, not long before his death. (Fig. 2.) When quite young he had a number of convulsions, from which he emerged paralyzed on the left side. He has ever since been subject to epileptic attacks, coming on at irregular intervals. His intelligence is very slight indeed; he is really imbecile. You will observe this mental deficiency from his facial expression. The paralyzed arm is the seat of a moderate contracture and deformity, especially so at the wrist. You will see a very marked disparity between the right and left side as to size. The left side has not developed as the right side, and it illustrates in a very decided manner the lack of development which occurs in the paralyzed parts in a large proportion of these cases. The difference between the two shoulders is not only manifest in the muscular development, but also in the size of the bony and ligamentous structures. The muscles of the arm and forearm also present the same marked difference. At the time the photograph was taken he was suffering from advanced phthisis.

You now have from these three cases a pretty complete clinical history of this class of cases. Few, if any, premonitory symptoms, a convulsion or series of convulsions, upon recovery from which the child



FIG. 1.—Right hemiplegia with contracture: case II.



FIG. 2.—Left hemiplegia showing marked disparity between the two sides: case III.

is found paralyzed upon one side. This condition may come on any time after birth up to the tenth or twelfth year of age, but it is most common from birth to the fifth or sixth year of age. The hemiplegia is permanent. Epileptic attacks often follow at intervals for years, and cease as the child grows older. In other cases the epilepsy only comes on years after the hemiplegia. In a few cases epilepsy does not occur.

There is a wide difference as to the amount of arrest of development in the paralyzed parts, as you see from the cases I have shown you. The same holds good for the contracture: the paralyzed muscles may be quite flaccid or there may be contracture of varying degree. The amount of mental defect also varies very much: there is always some lack of mental development up to decided imbecility. If the disease begins very early in life, there is an arrest of mental development. The frequency of the epileptic attacks also interferes with the development of the mind. Some small children have these seizures very frequently. In addition to the tremor which you have seen in the young man first shown you, there may also be present in some of these cases a peculiar spasmodic condition of the fingers and hand, which is known as athetosis; you will remember my having shown you a case of this kind early in this course of lectures. These tremors following after hemiplegias have often been called post-hemiplegic chorea or post-hemiplegic trembling.

The diagnosis is not difficult after the hemiplegia is established, the sudden appearance of the convulsions followed by a permanent hemiplegia. In tumor there are premonitory symptoms, such as headache, vomiting, optic neuritis, perhaps an increase in the size of the head, if the child is very young. If hemiplegia occurs, it is gradual in its onset and preceded for a considerable time by these above-mentioned symptoms. The other conditions mentioned are so rare that they hardly ever come in to render a diagnosis difficult.

The pathology in these cases is a more or less localized cerebral pathology with or without porencephalus.

I am able to show you brains illustrating these gross changes. First of all let me show the brain in the case of the young man whose photograph I showed you. Owing to the slow death from phthisis the brain was quite soft when removed, and on hardening does not show the various parts as well as if it were more firm on removal from the calvarium. You see the extreme atrophy of the right hemisphere; the cerebellum and corpora quadrigemina are entirely uncovered, owing to the extreme atrophy of the posterior part of the hemisphere. The tip end of the frontal lobe alone shows any evidence of the convolutions.

The cuts on the left hemisphere were made in order to preserve the brain. (Fig. 3.)

This second brain is taken from a girl of nineteen with left hemiplegia, moderate contractures in the paralyzed limbs, epilepsy, and imbecility. The position in which I show the brain is that which it occupied in the cranial cavity, which was small. You will observe from this view the marked atrophy of the right hemisphere and of its convolutions; the right lobe of the cerebellum is exposed in consequence. The left hemisphere is pushed over as it were to fill the space in the cranial cavity left vacant by the atrophy of the right hemisphere. (Fig. 4.)

I now show you the under surface of the same brain, and you again see the extreme atrophy of the right hemisphere. (Fig. 5.) You will also observe the decided atrophy of the right half of the pons, and please to observe that the left lobe of the cerebellum is very much smaller than the right. This is due to the degeneration or atrophy of the peduncular and other fibres which connect the cerebrum, pons, and cerebellum together, and of the pyramidal tract which passes into the medulla oblongata as the anterior pyramids and into the spinal cord as the direct and crossed pyramidal tracts.

This brain is from a boy nine years of age who, when an infant, fell out of a window, receiving a severe injury to his head. He is at present the subject of epilepsy and left hemiplegia with slight contracture. An examination of his head shows a considerable absence of bone; the opening was covered with a dense fibrous tissue; the opening in the bone is about the size of a fifty-cent silver piece. An operation was done with the hope of relieving the epilepsy. You will observe on the brain which I show you the decided loss of substance, extending the entire length of the brain posterior to the fissure of Rolando. The convolutions of this hemisphere are otherwise fully developed. The boy was intelligent and gave an account of himself. (Fig. 6.)

These specimens show the most common macroscopic changes observed in this class of cases. I will not to-day speak of the histologic changes which may be found in the brains of these children except in a general way. The largest proportion of these cases appear to be due primarily to a meningeal hemorrhage more or less extensive, which explains the sudden convulsive seizures in these patients. Subsequently an atrophy of the cortex, more or less extensive, follows. This is most probably the condition which has taken place in two of the brains which I have shown you. Traumatism giving rise to hemorrhage also gives a similar grouping of symptoms, as is illustrated in the last



FIG. 3.—Brain from case III.



FIG. 4.—Coronal aspect of the brain in case IV.



FIG. 5.—Base of the brain in case IV.



FIG. 6.—Lateral view of the brain from case V.

brain shown you, with the extensive loss of brain-substance. You must not forget that in all these cases when hemorrhage has occurred a certain amount of encephalitis follows; this adds to the disorganization of the nervous tissue. It has been claimed that some of these cases are due to an acute polioencephalitis, analogous to the acute poliomyelitis which is so common in children. I will admit that such a condition is possible, but at present our knowledge is largely confined to hemorrhage as the cause of these localized atrophies, or, as is believed by some, to occlusion of vessels,—embolism or thrombosis.

Treatment of these cases must consist of the ordinary treatment of convulsions, if you are called to see the child when it is in the convulsions. After the hemiplegia is established there is no specific treatment of these cases. The epilepsy which is apt to follow must be treated as epilepsy, by the moderate use of bromide of sodium. It is unwise to push the dose up to a large amount in these cases, as is sometimes necessary in idiopathic epilepsy.

Recently operation has been suggested in these cases. One can readily see from the specimens which I have shown you how hopeless such a procedure must be, and if the operation were done very early the result would be no better. In the case of the boy whose brain I showed you last an operation was done. This was easy, as there was an absence of bone; the space being covered with a dense fibrous tissue, into which an opening was easily made; a large quantity of clear serous fluid was discharged; this fluid was undoubtedly cerebro-spinal. A cavity was found which probably communicated with the lateral ventricle. The boy recovered from the operation. There was a draining of fluid from the opening for about one week; he had recovered sufficiently to be up and about the ward. One day he suddenly became comatose and died, probably as the result of the constant drain of cerebro-spinal fluid.

The contractures which follow in the paralyzed limbs may be treated by massage and electricity, to prevent the increase of this muscular rigidity. It does not cure the hemiplegia, as that is due to the injury by disease of the motor path to the brain.

ACUTE ANTERIOR POLIOMYELITIS.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC.

BY WILLIAM BROADDUS PRITCHARD, M.D.,

Adjunct Professor of Mental and Nervous Diseases in the New York Polyclinic.

GENTLEMEN,—I have had during the past three weeks the opportunity of showing you several types of acute and chronic disease of the spinal cord, including traumatic myelitis, syphilitic meningo-myelitis, locomotor ataxia, and chronic diffuse myelitis, so called. The clinic to-day is intended to illustrate still another form of disease of the spinal cord, probably the most common of all, chiefly affecting children, and known as poliomyelitis anterior acuta, or, as it is also termed, infantile spinal paralysis. It is an exceedingly interesting and important type for several reasons. As I have said, it is quite common, recognizing no climatic or social restrictions; its pathology is absolutely definite in the most important particulars, and the elements of uncertainty in the prognosis and the results of treatment are sufficient alone to stimulate a lively and continued interest, especially in individual cases. These four patients before you have been selected from a large number in attendance upon the clinic as representing various typical, as well as anomalous, phases of the affection. Before proceeding to demonstrate to you the points of interest connected with these individual cases, I shall endeavor to give you a symptom picture of the classical disease in order that you may have a clear mental standard of comparison. The disease is one of childhood in more than ninety per cent. of all cases, and it is the infantile form alone which we will consider to-day. The most frequent age of onset is between the second and fourth years. Its mode of onset is threefold. The paralysis, which, by the way, is the prominent and all-important symptom, may appear suddenly and without premonitory or coincident symptoms or warning of any kind; a child going to bed apparently perfectly well may be found the following morning to have lost the use of one or more limbs, or a part of a limb, while otherwise it is in the best of health. This type has been

designated West's morning paralysis. In other cases the invasion stage is characterized by fever, which varies in duration and degree and is not accompanied by other symptoms, as a rule, which indicate its nature or the results which are to follow. Occasionally this fever is accompanied with one or more convulsions. In cases preceded by convulsions, the convulsive seizure is usually unique, rarely recurring more than once or twice, and it is worthy of mention that the convulsive movements have often been observed to affect most severely the muscles which are afterwards to be paralyzed. When the paralysis is introduced by a fever this is usually of short duration and relatively mild degree, the fever disappearing abruptly upon the onset of the paralysis. We have very little positive knowledge of the etiology involved. The element of heredity plays a minor rôle. Trauma is a factor in some cases, and probably in many more than is supposed, the fact being difficult of proof for obvious reasons. The theory of an infectious origin has much to support it in the fact that many cases are observed as resulting in some way not definitely known from the infectious diseases, such as measles, scarlet fever, and diphtheria. The severity of the stage of invasion bears no definite relationship to the extent or completeness of the motor impairment which may occur, nor are there any other data by which this can be foretold. Certain general facts are, however, true of all cases. It is exceedingly rare in poliomyelitis to find the paralysis of hemiplegic distribution. Involvement of both upper extremities is also exceedingly uncommon, though paraplegia is not infrequently observed. The most common type is a partial or complete monoplegia of the leg or arm. Seguin, Gray, and others contend that the paralysis in its onset is probably general (diplegic) in all cases, though this stage is so short as to often escape attention. It should be borne in mind that the paralysis is always greatest at first, gradually receding spontaneously, although always leaving behind some permanently disabled muscle or group of muscles. The paralysis is always of the flaccid type, never spastic, the limb often hanging loosely from the body. The muscles affected in groups are invariably associated in function, as flexors, extensors, elevators, etc., although all groups may be sometimes involved in a limb. The muscles of the head and face are never involved. The one exception with which I am familiar is the case reported by Seguin, in which, with the deltoid group, there was paralysis of the temporal muscle of the same side. The deep reflexes are lost in poliomyelitis if their integrity is involved at all. The muscles begin to rapidly atrophy. *Contractures* do not occur, but *contractions* and deformities do. There are rarely any sensory

symptoms, although the limb affected is colder to the touch than its fellow, looks blue or white, and is of subnormal surface temperature as a rule. Following the atrophy, or preceding it, there is noticed an abnormal condition in the quantitative and qualitative degree to which the paralyzed muscles respond to electric stimulation. Muscular contractions in response to faradic stimulation are early lessened or lost. The same may occur to galvanic stimulation; and in addition a change occurs in the order of sequence in which a normal nerve or muscle responds to galvanism according as the positive or negative opening or closing current is used. This alteration of the normal qualitative electrical reaction is known as the reaction of degeneration. Two points of negative interest and importance exist in the absolute absence of tremor of any type in poliomyelitis and the absence of mental impairment. Having given you thus briefly a summary of the salient symptomatic and diagnostic points, we will proceed to an analysis of these examples of the disease before you.

The history of this boy on the right, whom we will designate Case I., is as follows: D. K., aged twenty-six months, born of healthy parents at full term and with normal labor, is one of seven children, all the rest of whom are healthy. Until he was twenty-one months old this child was healthy. His present trouble was first noticed by his mother, who found on dressing him one morning that his right arm was limp and powerless. The child was well the night previous; there was no fever, no convulsion, and no history of trauma. The patient seemed well and in good spirits in every other respect, not complaining then or since of any pain. This is the history of the development of the disease in this case, corresponding to the type referred to as West's morning paralysis. He was first brought to the clinic three months ago, just three weeks after the onset of paralysis. Examination at that time showed a well-developed, intelligent child with paralysis of the right arm, presenting much the same appearance which you see to-day. You will notice the bright, sensible expression of this child, the flabby, flaccid, limp position and appearance of the arm, the wasted condition of the right as compared with the left arm, the bluish-white color; and if you take an arm in each hand you notice at once that the right feels colder. With this pin I find that the child is normally sensitive to a slight prick, and with these two test-tubes, one filled with cold and the other with hot water, I test the sense of temperature, finding it intact. Tactile sense seems to be preserved, although difficult to test in one so young, while muscular sense we cannot test satisfactorily at all in young children. There is no involvement of any cranial nerve, as the

vision, hearing, taste, smell, speech, and facial movements and sensations are normal. I shall not have time to demonstrate the electrical changes present in this case before you. Tests have been repeatedly made, and the response to both faradic and galvanic currents found invariably abnormal. In fact, to the faradic there is scarcely any response at all, while with the galvanic there is an alteration of the normal polar formula of Erb, indicating degeneration in the nerve. You have in this case, then, all the salient symptoms I have mentioned, and the diagnosis is easily made. The only source of possible confusion which I can think of in this case is the elimination of neuritis of the brachial plexus or spinal hemorrhage. The latter is quite rare, is almost certain to be followed by graver symptoms often ending quickly in death, and to present mixed sensory and motor paralysis with bed-sores, etc., if the patient survive. In a neuritis there would be, as in this patient, a flaccid paralysis with atrophy and altered electrical reactions, but there would also be sensory phenomena, both subjective and objective. The child would feel pain, and at first there would be hyperæsthesia, followed later by loss of pain, tactile, and, perhaps, temperature sense. The mode of onset would be much more gradual, and usually, if not always in young children, neuritis is due to trauma. Other symptoms which are present in adult cases of neuritis, such as tremor, paræsthesia, etc., are not observed in young children and cannot be relied upon, therefore, in a differential diagnosis.

CASE II.—This little girl has the following history: M. F., aged nine years; residence, Long Island. Family history negative. Healthy until five years of age, when she had an attack of fever lasting five days, diagnosed as malaria, but which was followed by a paralysis which at first affected both legs. In a day or two she recovered the full use of the right leg, but the left from the knee down was absolutely powerless and “dangled” limp and flaccid. She complained of no pain, and after the paralysis had no more fever, and except for the paralysis was as well as ever in one week. You notice in her an equally intelligent face, an absence of cranial nerve involvement, and I show you the same absence of any sensory impairment in the leg. On measuring the two limbs I find the left calf one inch smaller than the right, and at one inch above the ankle a proportionate diminution in size. On testing the knee jerks I find the left entirely gone, while the right is only slight even under reinforcement. The foot looks blue and feels colder than its fellow; it hangs forward and shows one of the deformities which occur so often in these cases,—that of an equinus. Notice the

absence of any spastic rigidity, contracture, or clonus in this case, a point of value in excluding a cerebral origin.

CASE III.—M. S., three years old ; German parents. Family history negative. Born at full term, normal labor. Well until two years and two months old, when he was attacked by a fever which lasted two days and seemed slight, but which was accompanied by two convulsions, some vomiting, and great weakness. In a day or two he seemed well again, but could not use his left arm. Some power gradually returned in the arm, especially in the hand and forearm, but at the end of two weeks, when brought to the clinic, he had lost absolutely the power to elevate the arm at the shoulder. There was evidence of a beginning atrophy of the shoulder muscles, the deltoid, coraco-brachialis, and biceps ; and the response to faradism was sluggish and imperfect as compared with the right arm. The test for the electrical reactions in these cases should, by the way, always be made of both the sound and the affected limb, as a defect is brought out more plainly by comparison. The boy, as you see, is a healthy, bright-looking little fellow, "very smart," as his mother truthfully says, talking well, with a good memory, and showing no defect in any cranial nerve function. The two shoulders do not show as much difference as they did some months ago, as the atrophy has been arrested by treatment. It is, however, quite apparent in the flattening of the left shoulder. When I ask him to raise the two arms as high as he can, you notice that the right goes up to the perpendicular, while the left gets no higher than a position representing an angle of about forty-five degrees with the floor. There was no sensory impairment in this case. The electrical reactions in the circumflex nerve in this case are much nearer the normal than they have been, but there are still slight qualitative changes.

CASE IV.—M. M., aged ten years ; never been very strong, though never showed any nervous symptoms. Good family history. One year ago had scarlatina quite badly, but without delirium or convulsions. During convalescence it was found that he had lost the use of his lower limbs. No rectal or vesical symptoms, no pain or defect of sensation, no mental impairment. Slowly he regained some power in his lower limbs, and at the end of two weeks could move his limbs about in bed to a certain extent, but could not walk or stand on them. He was treated for two months at home, an orthopædic brace applied, and with this assistance and his father's support he could walk a little. When examined he was found to have a flaccid paraplegia, both limbs cold and blue, both knee jerks abolished. No defect of tactile, pain, muscular, or temperature sense, no rectal or vesical symptoms, no tro-

phic skin affections. Both legs were atrophied, especially the calf muscles and the anterior tibial group. There was total loss of faradic excitability with any current which the patient would stand, while galvanism showed the reaction of degeneration in the anterior and posterior tibial and peroneal groups. This boy studies at home, learns well, and is much improved as to his paralysis, having been under continuous treatment with us for nearly a year.

I have shown you in these four cases a leg type, an arm type, a shoulder type, and a paraplegic type of the disease. I have shown you cases beginning with fever, a case with initial convulsions, a case following an infectious disease, and one of the West's morning type. The etiology may vary, and so may the distribution and degree of paralysis; but the essential pathology is always the same,—that of a destructive disease of the larger or giant cells of the anterior horns of the spinal cord. The nerves may and do take on secondary disease, and the muscles secondarily atrophy, but the primary lesion is of these cells, as indicated in the name of the disease. The prognosis and treatment are also well illustrated in these four cases. Cases I. and II. have shown only slight improvement, although treated according to the best methods generally accepted, and which I shall mention in detail. Case III. has made excellent progress towards recovery, but there will very probably always be some evidence of the disease. The case of paraplegia which I showed you in the last patient, has improved very much and will improve more. The treatment in all of these cases has been the same. They have all had from one to three applications of galvanism or faradism every week, and, as the acute stage had passed in each case when seen, each patient has been given strychnia in doses varying with the age. In private practice I should recommend that the strychnia be given hypodermically, as it seems to exert a more prompt and better effect. Small doses of potassium iodide have been given to each of these patients daily. The mothers have been directed to practise massage, rubbing the paralyzed limb once or twice daily. In using electricity in these cases, we begin, if possible, within a week or ten days after the onset, using first the continuous galvanic current, followed in a few days or weeks with the interrupted galvanic, using from five to ten milliamperes of current for ten or fifteen minutes. One electrode, a large flat sponge well moistened, should be placed over the spine at a point corresponding to the location of the lesion; the other, a smaller sponge or cloth-covered electrode, should be placed upon the limb affected. As soon as faradic contractions are established, this current should be used alternately with the galvanic. Various mechanical ap-

pliances may be made to do adjunct service in promoting nutrition and strength of a compensatory character in muscles which are not paralyzed. In the leg cases I have used the tricycle with good effect. These measures comprehend our most reliable therapeutic resources, but with the most energetic and prompt treatment the result, so far as the permanent residual paralysis is concerned, is often most disappointing. No case should be neglected, however, as it is impossible to foretell the result in individual examples of the disease. Some approximate idea may, however, be gained in certain cases, the extent of the paralysis and the degree of electrical change, with rapidity and extent of the atrophy, constituting data upon which to make a fairly accurate prognosis. I have said nothing as to the treatment in the acute or invasion stage, for the simple reason that it will be so seldom recognized as to make such reference comparatively useless. Ice or counter-irritants to the spine, with full doses of ergot and small doses of quinine, are the measures advocated in the text-books for this stage when recognized.

Surgery.

A CASE OF CONGENITAL TUMOR ON THE LEFT FOOT.

CLINICAL LECTURE DELIVERED AT THE MIDDLESEX HOSPITAL.

BY J. W. HULKE, F.R.S., F.R.C.S.,

President of the Royal College of Surgeons of England; Senior Surgeon to the Middlesex Hospital.

GENTLEMEN,—The subject of my remarks to-day is a little boy who has been under your observation during several weeks in Percy Ward, and is now about to return to his home in the Isle of Wight. He was sent up to me by my former dresser, Dr. Hollis, who has for several years practised at Freshwater. Eight years old, our little patient is a rather puny, pale, delicate-looking child, with a worn, suffering expression of face. On the upper surface of his left foot, extending from a little above the bend of the ankle to within about two finger-breadths of the root of the toes, is a conspicuously prominent swelling, the convex surface of which is broken by deep furrows into a few large transverse lobes and some smaller lobules, a configuration manifestly impressed on the swelling by the movements of the ankle-joint in flexion and extension. The consistence of the swelling varies greatly; in parts it is as soft and elastic as a myxoma; in parts it has the firmness of a soft fibroma; scattered through it are small, hard pellets suggesting thrombi or even phlebolites; and over the bases of the first and second metatarsal bones are hard masses of uneven figure such as might be caused by hyperostoses. The integument corresponding to the swelling is of a dull red color, and it is overrun by a net of dilated veinlets. It cannot be pinched up in a fold, but its under surface seems to merge into the tissues of the underlying swelling. The swelling is so tender that the child cries when it is handled, and it is also very painful. He cannot bear the pressure of his boot. "His foot," his mother says, "has hurt him so much that he has not been able to play." Above this swelling, in the course of the musculo-cutaneous nerve, reaching upwards from the bend of the ankle to the middle of the leg, a coarsely-

beaded subcutaneous cord can be felt, over which the skin is freely movable and of normal appearance. From his mother we learned that the swelling was present at birth, that its color in early infancy was "scarlet,"



and that it did not seem to be painful till the child "got its shoes." She further told us that "she laid it" to an unsatisfied "longing for a sole" (*Pleuronectes solea*, Linn.), and she explained that "whilst carrying this child" she had one day an intense longing for a sole; a fisherman brought a basket of these fish to her door for sale, but as he and she could not agree about the price he would not sell, and she went without the sole for which she craved. So when the child was born and the swelling on its foot was seen, the explanation of the prodigy was to her mind plain enough, the swelling was the "mark of the sole;" and, as if this were not enough mischief to have accrued from

an unsatisfied longing, she added that she could see a faint mark of a sole on the boy's right foot and also on each foot of his twin brother. The mark on the child's right foot to which she now called our attention was to our incredulous eyes the swelling formed by the belly of the extensor brevis muscle, and Dr. Hollis, who examined for me the twin brother's feet, wrote to me that here also this same muscle was the mother's "sole-mark." At the time of the occurrence of this profound impression upon her, her legs and feet were, she said, swollen and hard; she afterwards laid herself on a sofa and rubbed them. Her opinion was that if she could have had the sole, the child would not have had the swelling.

And now, perhaps, some of you are wondering why I have wasted your time with this "old wives' fable." I am not so certain that I have really wasted your time by recounting to you this curious instance of *folk-lore*, which, although in this nineteenth century and in the better

educated classes appears to be dying out, is not restricted to any class, is not limited to any country, which reaches back to the very earliest times of which we possess a written record, and which probably in those patriarchal days was no new idea, as the account of the device in the matter of sheep-breeding employed by Jacob with a design for his own advantage plainly tells us in the book of *Genesis*. But perhaps some of you would like to ask me, is the existence of such an influence as "maternal impression" credible? To this inquiry it is difficult to give a direct answer,—to reply simply yes or no. The great majority of alleged instances on which this belief rests certainly break down when investigated. This happened in our present supposed example as regards the boy's right foot, and also his brother's feet, where the ignorant mother, "disposed to see," saw the "ghosts" of soles in that which we recognized as a natural structure. Then as to the right foot, the mother's craving and her disappointment: unless she designedly told us a fiction, which I have not any reason to suppose, this occurred when she was already six months pregnant. Those who are less sceptical as to the validity of the doctrine of maternal impressions influencing the embryo in the way of causing any of the multifarious phenomena comprised under "mother's marks," appear to concur in the opinion that the circumstance is one incidental to the early phase of embryonic existence; whereas, in the present alleged instance, the foetus had attained already so high a degree of development that some would even regard it as possibly viable and capable of independent existence if suitably cared for. The very late period in foetal life when this circumstance happened (if it happened) justifies us, I think, in considering the association of his mother's "disappointed craving" and the swelling on her child's foot as merely accidental; it should be regarded as a coincidence not more curious than many others of frequent occurrence. There remain a very few, a vanishing number of instances which may not be so easily dismissed. Even these few would possibly, or to put more forcibly, would probably break down if the evidence on which they rest could be closely and carefully examined. Let me, however, stay from treading further the maze of this digression and come back to the matter-of-fact question,—the nature of the tumor. The rough, objective, clinical characters of the bulk of the mass were those of a soft variety of that which Professor Virchow many years ago designated a diffuse fibroma. Good examples of this are presented by some instances of spurious elephantiasis. Not a few of these have an inflammatory origin; the enlargement is first noticed as concurrent with, or as consecutive upon, a dermatitis of a mild erysipelatous type. This

is prone to recur, and each attack brings with it a fresh accession to the mass. Beginning in the skin, the process subsequently involves all the connective substances, and when occurring in a limb it in time implicates even the bones, leading to irregular overgrowth of these. In our present case the swelling began in utero, which made such inflammatory origin highly improbable, but the mother's account of the progress since birth was consistent with the idea that fluxionary congestion attending slight inflammation might have been a factor in the increase of the mass. What were the hard knots scattered through the mass? Considered together with the net of cutaneous veins and the lobulation of the mass, I was inclined to think they might be venous thrombi or, perhaps, even vein-stones. If so, then there was probability that the swelling was primitively a venous nævus, and its present condition was due to retrogressive changes. For these scattered knots, however, there was also another explanation, the key to which was the beaded cord in the course of the musculo-cutaneous nerve in the leg, for this, with much probability, indicated neuroma. I need scarcely remind you that neuromata are of at least two kinds, those in which the bulk of the tumor consists of nerve-tissue,—of these from personal research my knowledge is very limited,—and those in which some one of the forms of connective substance constitutes the principal bulk; examples of this latter sub-class are not very infrequent. As illustrations I might cite those exquisitely painful little subcutaneous knots of which early in this century Dr. Wood gave a good description, for at least some of these are false neuromata. Here also belong those bulbous swellings of the ends of nerve-trunks in amputation stumps, the presence of which is accompanied, in some instances, with very acute suffering; and here, too, belong those very remarkable multiple neuromata implicating extensively the spinal nerves, of which our museum possesses such admirable examples. (Cat., Nos. 884, 885, 862.) Our provisional diagnosis was that the tumor, in respect of the tissues composing its principal bulk, was a fibro-myxoma, a supposition which best accorded with its congenital origin and its objective characters, that it might comprise degenerating nævoid tissue and possibly neuromata.

Removal of the tumor offered, obviously, the only prospect of relief, and, his mother acquiescing, the principal mass was excised on the 1st of November. The blending of the deeper layers of the skin with the tumor made it impracticable to cleanly detach it from the latter and turn back flaps composed of skin only, the reflection of a thin layer of the tumor-tissue with the skin was unavoidable, but the tumor itself was separable without difficulty from the deep fascia. In the course of the

dissection it became apparent that the small, hard knots which had simulated thrombi or phlebolites were neuromata grouped along the branches of the musculo-cutaneous nerve, so numerous as to form small beaded bundles. Of these a few larger than the others composed the firm uneven knots overlying the bases of the first and second metatarsal bones, which had simulated hyperostoses of these bones. On the anterior tibial nerve passing to the first interdigital cleft, and on the twigs of the long saphena nerve coursing along the inner border of the foot, recognized in the dissection, no neuromata were found. After the removal of the main part of the mass on the foot the incision was prolonged up the leg, over the beaded cord felt there, and this was dissected out. It proved to be a string of fusiform swellings on the musculo-cutaneous nerve. Redundant portions of the flaps having been removed, the wounds were closed and the foot and leg fixed on a splint. The operation was attended with but slight bleeding. Immediate union of the greater part of the wound was obtained. Slight sloughing occurred near the roots of the toes. On the 28th of December, and on the 17th of January last, some small lobules not comprised in the first operation were excised. On both these occasions immediate union occurred. On February 15 he was fetched home by his mother, who was delighted to find him merry and bright, very different from the moping, suffering child she had brought in October to the hospital. It should be added that the skin over the instep remains thick, and it wants the normal suppleness and extensibility so characteristic of the child's skin. I would also call your attention to a fact not without significance. It is that the scar of the incision in the leg has assumed a cheloid character.

Sections of the neuromata made by our surgical registrar, Mr. Murray, show bundles of nerve-tubules widely separated by a large quantity of interstitial connective substance, the bulk of which largely preponderates over the nervous elements. The mass of the tumor also consists of connective substance, characterized by a very large admixture of the corpuscular element and only sparingly vascular. Structurally, then, the tumor appears to come within the category of *elephantiasis mollis congenita* of older clinicians, in which the enlargement has frequently a relatively restricted extent. The apparent limitation of the neuromatous condition to the musculo-cutaneous nerve, and the escape of the anterior tibial and the long saphena nerves in the foot, is a noteworthy circumstance. The pathological process occasioning the nerve-knots I suppose we may regard as a neuritis. I have sometimes fancied that in racemose or cirroid aneurism we have an analogy in the arterial system.

**REAMPUTATION—SUPRACONDYLOID—OF THIGH;
BURSITIS OF BURSA IN FRONT OF TUBERCLE
OF TIBIA; RECURRING OSTEO-MYELITIS (TU-
BERCULAR); TUBERCULAR ABSCESSSES OF THO-
RACIC WALL.**

CLINICAL LECTURE DELIVERED AT THE BUFFALO GENERAL HOSPITAL.

BY ROSWELL PARK, M.D.,

Professor of Surgery, University of Buffalo, Medical Department; Surgeon to Buf-
falo General Hospital.

GENTLEMEN,—Three weeks and a half ago a young man was injured in a railway accident in a neighboring town, and he sustained an amputation of the leg near the knee. The condition of the leg now is such as to demand reamputation. It may be the fault of the doctor who performed the first operation, or it may be in no sense his fault. The patient was brought here two weeks after the amputation, with a stump whose flaps were sloughing badly all around. That accident happens at times under the most favorable circumstances, and, while the condition is a deplorable one, it does not necessarily reflect discredit on the operator.

The stump is about three and a half inches below the knee. The skin flaps have entirely sloughed away, leaving the muscles exposed and at present nicely granulating. At the time of the patient's entrance, however, the condition was one of suppuration and foulness, but by the use of hydrogen peroxide sprays and other antiseptic treatment the putrefactive element has been eliminated. But under the most favorable circumstances cicatrization would only occur slowly, requiring three months or more of hospital life, and leaving the patient with a stump uncovered by true skin and not fitted to support any weight. The patient has, therefore, at my suggestion, preferred to submit to reamputation rather than to endure so long a delay, that by the operation he may secure a useful stump.

I am compelled by the lack of tissue to amputate at any rate as high as the knee-joint. Such stumps turn out fairly well and afford a useful basis for the application of artificial limbs, but there is an im-

provement on this operation at the knee-joint which I shall probably carry out in this instance. If the amputation be made at the joint proper, the patella may be left or removed at the fancy of the operator, but there is no fresh bony surface to hold the patella in place if it be left. The patella is virtually the knee, as we use it for resting purposes, and it is provided with a bursa with pretty tough skin in front of it, so that it is possible with practice—though I am afraid that few of us get it—to spend considerable time on the knees without discomfort. If this skin, bursa, and bone can be retained, we have really a very serviceable kind of stump, and this end can be attained in the following manner: We can saw off the lower extremity of the femur and the posterior surface of the patella, and approximate the two raw bony surfaces. This is known as the supracondyloid or Gritti's amputation. Accurately speaking, the amputation should be called transcondyloid, for it is through and not above the condyles. It was Mr. Stokes, of the Dublin Hospital, who suggested the supracondyloid operation proper. If we cut the femur across at Gritti's line, the femoral surface is larger than the patellar, and the coaptation is not good. I shall probably use Stokes's operation, therefore, which allows a nicer coaptation of the two bone surfaces. This leaves a little shorter stump than the knee-joint amputation, but the skin over the end of the stump after the latter operation is thin, and it will not bear much pressure without ulcerating. This is not a great inconvenience, because artificial limbs are made so as to distribute the pressure over the sides of the stump. It is well, however, to have the skin tough and hard, so that weight may be borne directly on the end of the stump if necessary.

[The patient was brought in under the influence of ether, with the knee covered with an antiseptic dressing. The Esmarch bandage was applied above this, so as to avoid forcing any infectious matter into the absorbent surface of the stump.]

The habitual position of the stump when the amputation has been high up in the leg, is one of flexion, as you see illustrated here. This can be overcome by tenotomy of the hamstring tendons, or by dressing the stump on a splint; but, unless you have the forethought to observe these precautions, the result is not good.

[After cleansing and shaving the thigh, it was washed with sublimate solution, and the operation performed, the instruments, previously boiled, being taken from a saturated, watery solution of hydronaphthol. The anterior incision was made from without inward, the posterior by the transfixion knife from within outward. The femur was sawed

through above the condyles ; the patella was then held in the Farrabœuf long-beaked forceps, and its posterior surface removed with the saw. The popliteal nerves were pulled down and cut off. The popliteal artery and vein were tied in one large catgut ligature. Dr. Park remarked that it was theoretically better to tie the vein and artery separately, but that he knew of no bad results from tying them together. He continued :]

In the good old days when I was a student, studying what was supposed to be surgery, all my teachers tied these vessels with dirty silk, and left long ends hanging out, and after about two weeks, at each dressing they would pull on the ligature, about as one would on a bell-cord, to see if it was ready to pull off. This was the result of the use of septic and non-absorbable material, though aseptic silk will be absorbed. Another advantage of this operation over that at the knee-joint is that we do not have so many vessels to tie. The freshened bone surface of the patella is sewed to the end of the femur by a couple of silkworm-gut sutures passed through the soft tissues. I suture the deeper tissues of the stump with buried sutures of catgut in order to leave less of a "dead-space" for blood to collect in, and thus to hasten healing. No drainage-tube is inserted, for the wound is aseptic, and gravity will remove all the fluid that may collect. The skin is anointed and dusted with iodoform, and Lister's new antiseptic gauze, impregnated with the cyanide of zinc and mercury, is applied. This new antiseptic has no advantage over bichloride of mercury in antiseptic power, but it is less irritating. The dressing will not be removed for ten days or two weeks, unless some bad symptom arises. This is quite an improvement on the old method. When I was a hospital interne we had to dress these stumps morning and night.

[Perfect union without pus.]

BURSITIS OF BURSA IN FRONT OF TUBERCLE OF TIBIA.

Here is a man of forty-one, who says he fell and hurt the upper part of his leg a year ago. Within the past four weeks a little swelling has appeared just below the patella. It fluctuates and it is somewhat painful. There is no *prima facie* reason why a man could not have an abscess here as well as in any other part of the body, and yet I do not think that such is the case here. This is an old injury. There are present swelling, redness, heat, soreness, and some loss of function. The patella is quite movable, and movement of it causes no pain. The swelling is about on a level with the tubercle of the tibia, not in front of the patella. The question is, What part

is involved? I must remind you of certain anatomical structures, the so-called bursæ or synovial pouches, which enable the skin to move over deeper parts, especially a prominent bone, or which allow a tendon like that of the patella or the tendo Achillis to move without friction over deeper parts. There are scores of these bursæ in different parts of the body at exposed places, to permit free play of one tissue or layer over another. There is one in front of the patella itself, and another over the tubercle of the tibia; these are usually not connected. This case is one of inflammation of the bursa over the tubercle of the tibia. There has been a collection of fluid there, in all probability serous at first, but which may have broken down into pus later. The house-surgeon made a puncture here a day or two ago and got, not pus, but thick serum, which settles the question between abscess and serous accumulation. A bursitis of this kind, followed sometimes by suppuration, but more often leading to a chronic thickening, on account of infiltration of the ordinary granular form, often occurs in late secondary or early tertiary syphilis. This may break down on the surface, making a ragged ulcer in front of the knee.

A prepatellar bursitis is ordinarily known as "house-maid's knee," and it is supposed to come from the constant irritation of that bursa in women who scrub. This is scarcely a case of house-maid's knee, not because the man is not a house-maid, but because his trouble is in a bursa lower down than that in front of the patella, but it is a trouble of the same nature. I think that nine cases out of ten of inflamed and ulcerated prepatellar bursæ are due to a local injury in syphilitic subjects. In this case there is no syphilitic element apparent.

I introduce a little cocaine under the skin and slit the tumor up freely. Sometimes these bursæ about the knee communicate with the joint, and so they must be handled with great care. As a rule, they do not connect with the joint, but it is well to proceed just as if every one of them did, for if any septic trouble began on the outside it would be communicated to the joint cavity. A good deal of thickened serum and some flocculi of fibrin are evacuated, and, with a small, sharp spoon, I scrape out the bursa. I could dissect out the sac, which would effect a theoretically perfect cure, but a simpler and equally effective method is to pack the sac with some material which shall allow it to heal at the deeper portions first, and thus to cause an obliteration of the bursa by adhesive inflammation. Cyanide of mercury and zinc gauze will be used, and in three or four days the wound will heal by "third intention,"—that is, by the union of two granulating surfaces.

RECURRING TUBERCULAR OSTEO-MYELITIS.

I have talked to you a number of times about the recurrence of tubercular and suppurative trouble in one bone after another, and the fact that pyogenic germs, especially staphylococci, may collect in little nests or conceal themselves in minute cavities in various bones and lie latent or idle for a number of years, and then apparently renew their activity. One of the best illustrations which I could have given you is the patient whom I presented at a previous clinic, and who had extensive caries necrotica. I operated on him three years ago, and at that time he gave the history that ever since he was a little child he had had abscesses in numerous parts of his body. Especially as a young man, he would develop abscesses here and there, the ordinary phenomena would obtain, and little particles of bone would be discharged. In his right arm, three years ago, and in his left humerus quite recently, there developed trouble of the same character, but excessive in extent and in degree. Those of you who saw the operation last Saturday will remember that the cavity in his humerus extended up almost to the upper joint extremity of the bone, the beginning of the process having occurred near the elbow. There was a large cavity, the bone having materially altered in shape and size, and its medullary cavity having expanded so that it would hold a section of a broom-stick without difficulty. This cavity was filled up with tubercular granulations, dead bone, and *débris*.

You know also how the tuberculous process may manifest itself here and there and everywhere, successively, and how it is possible to have repeated local tubercular invasions without reaching the point of general tuberculosis. Twice this winter I have had to operate before you on a young girl, the first time performing a resection of the hip, which was necessitated by an acute osteo-myelitis of the upper end of the femur, and I think that the osteo-myelitis was in this instance, so to speak, a galloping consumption of that particular bone. At about the time of this operation her right shoulder became sore, and, instead of getting better, she grew worse, and she came before you eight or ten weeks later, when I resected that joint. You remember that I withdrew from the end of the humerus a sequestrum fully five inches long. Since I operated on her right shoulder, which is now nearly well, she has complained of a sore spot in the middle of the left humerus, and of late this has become swollen and tender, and she now has undoubtedly a local tubercular focus at this point; and, therefore, inasmuch as it is getting worse, while her condition in other respects is

improving, I propose to cut down on the bone and try to remove the local disease. How much longer this programme will continue I cannot say. I can do little more for her general condition than by toning her up in every possible way with cod-liver oil and other tonics, and the internal use of guaiacol, and I am anxious to get her sufficiently well to go back into the country where she lives and where she can get more oxygen and more ozone than she can in a hospital. I am trying to conclude with surgery in her case and return to hygiene alone, but I seem to be handicapped by the development of these lesions. I do not think this is an extensive lesion in the arm, but it needs attention. I should not be surprised to find a little sequestrum in the bone.

I have exposed by incision only a small area of bone, but there is a little opening about the size of the end of a grooved director through which something is pouting out, which I suppose to be the tubercular mass trying to penetrate from the interior. As I enlarge the opening by using the mallet and chisel, a quantity of puruloid material of about the consistency of thin jelly, too thick to escape, but thin enough to project, appears, and here is a sequestrum which I have broken in removing a portion. After removing the rest of the dead bone I scrape out the wound and spray it with a sublimate solution and then pack it with antiseptic gauze.

This is what I should call technically a subacute tubercular osteomyelitis. We see a good deal of this kind of disease in this part of the country. If our patients were all Germans I should say that we operated under almost the same conditions as they do in some parts of Germany. In Göttingen, Professor König claims that thirty-three per cent. of his cases are tubercular. We do not get such a percentage here, but recollect the fact that not a week has gone by without your seeing surgical tuberculosis in one form or another, and in the medical clinics I presume you have seen the same disease in many of its protean forms.

SUBCOSTAL ABSCESS.

This next case is that of a young man of twenty, who has been sick for some time. He has had one abscess after another about the thorax and upper part of the body. I learn of no abscess that he has had below the waist. I shall be able to show you sinuses in two or three places, and perhaps the scars of two or three others. I only saw him in consultation a few days ago, and I advised the operation which I shall practise now. There is a series of deep, subfascial abscesses, accompanied by more or less retention of pus, and septicæmic symptoms have been thereby produced, the ultimate cause being unknown.

During the last few days his temperature has been running rather high, and he has had a continued fever. His evening temperature has averaged 103° , with morning remissions.

You see a sinus opening well down on the side of the thorax, and by pressure quite a quantity of pus can be forced out, which makes me think that underneath the skin and fascia, and probably underneath the pectoral muscles, there is a large flat abscess cavity. Whether this extends into the thoracic cavity or not I do not know, nor do I know what we shall find about the back. His high temperature is due to the retention of pus. There is a question as to whether he has miliary tuberculosis. Yesterday Dr. Rochester examined him carefully, and came to the conclusion that such was not the case, at least that he had not phthisis, although there was a little increased vocal fremitus and some dulness low down in the left side of the chest anteriorly. This probably indicates a little thickening of the pleural membrane, but it may indicate a localized tuberculosis of the lung. Or there may be a subcostal abscess between the costal pleura and the ribs themselves. It does not appear to be a case of empyema with spontaneous perforation, for there is no history of pleurisy, nor are there evidences of involvement of the thoracic cavity at present.

As the patient struggles under the anæsthetic, a quantity of pus is forced out of this sinus between the ribs, more than would be expected from an extrathoracic abscess. I will make several test-tube cultures of the pus.

[The chest was shaved and washed, and lastly douched with a mixture of turpentine and alcohol. The opening of the sinus was enlarged with the bistoury and the finger, a sound inserted, which passed upward six inches to a point about four inches above the right nipple. A counter-opening was made on the end of the sound. An incision, three inches in length, was made upward and inward through the soft tissues of the thoracic wall. Two or three intercostal vessels were caught with hæmostats. The walls of the abscess were then scraped with the sharp spoon. A sinus was discovered leading underneath the fifth rib. A piece of the rib was therefore resected with bone-forceps, and a considerable abscess-cavity was found outside the pleura but beneath the ribs. This was also curetted. It was found necessary to go through another rib, and the periosteum accordingly was split and pushed backward and the bone cut through. This abscess-cavity led up beneath the cartilages of the seventh and eighth ribs, nearly to the sternum.]

Having thus taken care of the suppurating tissue in front, we will

now turn the patient over and attack the posterior abscess. Proceeding here in the same general way, we first enlarge the sinus, which you see freely discharging at the tenth rib. It always pays to make a free opening for purposes of exploration, and you see here that this incision is between three and four inches long. I now find the large irregular cavity of an old tuberculous or cold abscess, and I proceed to scrape it out and cleanse it in the usual way. After having it pretty thoroughly scraped out, I find a sinus leading in beneath one of the floating ribs. As it is impossible to follow this to its termination otherwise, I remove a piece of one of these ribs in the same way as was done in front. I find now that the abscess extends some three inches upward towards the lower side of the diaphragm, and here it seems to come to an end. I have a suspicion that this cavity might be found to connect indirectly with the other, but the boy is too weak to justify so extensive an exploration at this time. After thoroughly cleansing and disinfecting both cavities with hydrogen peroxide, I pack them with gauze, not making the slightest effort to suture them together, since experience has shown that in such cases final healing is more expeditiously obtained by this kind of open treatment than by an endeavor to approximate the margins of the wounds.

[NOTE.—The boy's temperature went down, and locally the wounds improved day by day, but gradually the patient became weaker and weaker, and he succumbed on the sixth day after the operation, of sheer exhaustion, in spite of all efforts to sustain and stimulate him. There was no further sign nor appearance of sepsis.]

A CASE OF LACERATED WOUND OF THE CALF OF THE LEG, FOLLOWED BY SEPTIC GANGRENE.

CLINICAL LECTURE DELIVERED AT ST. GEORGE'S HOSPITAL.

BY T. PICKERING PICK, M.D.,

Surgeon to St. George's Hospital, etc.

GENTLEMEN,—I admitted into the hospital a short time ago a patient suffering from a severe lacerated wound of the calf of the leg. The patient was subsequently attacked with a form of gangrene which now, happily, is very rare, but which in my student days was much more common. I have thought that I should do well if I devoted the time at our disposal this afternoon to a consideration of this case. It is, as I have just said, nowadays a disease of rare occurrence, so that it is possible that during the time of your studentship here you may not have another opportunity of seeing such a case, and therefore it is most desirable that you should make the most of the chance which now offers, and this more especially because it is a disease which allows of no delay as regards treatment. It is a disease with which, should it ever be your fate to meet with such a case, you must be prepared to deal at once, if you would wish to rescue your patient from the deadly peril in which he is placed. There is no time for delay, no time for thinking over the case, no time for obtaining a second opinion, no time for consulting your text-books on the subject. No; you must be prepared to act at once and promptly, as the only chance of saving the life of your patient. Those of you who went round with me on the Monday on which I amputated this poor fellow's leg will remember that as soon as I saw the case I decided on immediate amputation, and that within an hour of my seeing the patient and recognizing his condition the man's leg was removed and he was back again in his bed.

The form of gangrene of which I am speaking has received many names,—*spreading gangrene*, *spreading traumatic gangrene*, *inflammatory gangrene*, *septic gangrene*; all to a certain extent express the condition. But of all the names, I think the one given to it by Maisonneuve is the best: "*gangrène foudroyante*," *terrible*, *dreadful*,

fulminating gangrene, indicates most accurately the nature of the disease.

I will first read you the notes of the case, as taken by the dresser, and then make such remarks as I can upon its etiology, symptoms, and treatment.

John T., aged forty, was admitted into Oxford Ward on April 21, 1893. He stated that whilst at work upon a crane he was struck upon the left leg by a heavy iron girder. When admitted he was collapsed from loss of blood. There was a large lacerated wound on the posterior and outer side of the left calf, which extended deeply into the muscles, so that the finger could be passed easily through the substance of the calf. There was a comminuted fracture of the fibula, but no fracture of the tibia. There was much bruising of the tissues around the wound. The wound was thoroughly irrigated with a twenty-per-cent. solution of carbolic acid; several loose fragments of bone were removed and one or two small bleeding vessels were tied. A drainage-tube was inserted, the wound dressed with double cyanide gauze, and the leg placed upon a splint.

On the following day there was found to have been some oozing, and the wound was dressed. It looked quiet, but the patient complained of much pain, and the temperature was 101.8° F. He was ordered a hypodermic injection of a third of a grain of morphia. Two days later (April 24) the temperature was 101.2° F. The bowels had been opened for the first time since the accident. The urine, hitherto normal, contained a slight trace of albumen; its specific gravity was 1024. The pulse was quick and feeble, and he was perspiring profusely, with an anxious expression of countenance. The wound in the calf was black and foul smelling. There was a dull-red or purplish-black discoloration extending up the inner and posterior aspect of the leg and lower part of the thigh, with much swelling and infiltration of the subcutaneous tissues and some emphysematous crackling in the neighborhood of the wound. Amputation was at once decided upon, and the limb was removed at the junction of the upper and middle thirds of the thigh, by a long anterior and short posterior flap. Owing to portions of the flaps, especially the posterior one, being infiltrated by a peculiar greenish, gelatinous material, they were trimmed, the gelatinous material being removed as far as possible; they were then scrubbed with a solution of zinc chloride (gr. xl. ad 3i), and the whole stump well irrigated with a solution of hot corrosive sublimate (1 in 500). Two large drainage-tubes were inserted, and the wound closed with silver sutures and dressed with cyanide gauze.

He was ordered a hypodermic injection of morphia (gr. $\frac{1}{2}$) every six hours, six ounces of brandy daily, with as much fluid nourishment in the shape of beef-tea and milk as he could take.

I will not weary you with a recital of the daily record of the case. Suffice it to say that for some days the patient's condition was critical. The pulse was quick, feeble, and at one time intermittent. The temperature ranged from 100° to 101° F., and was never as high as 102° F. There was some sloughing of the stump, and foul pultaceous masses separated in spite of a daily irrigation with a strong solution of corrosive sublimate, but through it all he took his food freely, and though, according to his own statement, a total abstainer, showed a particular predilection for his stimulants. He is now nearly well, and will leave the hospital for our convalescent branch at Wimbledon in a few days.

I will ask you to note, in connection with the case I have just read, that the original wound in which the disease commenced was a very irregular and lacerated one of large size, with, no doubt, many outlying pockets or recesses from which it would be exceedingly difficult to get rid of any blood or serum or other putrescible animal matter. I will also ask you to note that after the amputation and the removal of the gangrenous parts there was no further spread of the disease. It is true there was some sloughing of the tissues in the neighborhood of the wound, the reason of which I will explain to you immediately; but this was merely local gangrene. There was no further spreading, and therefore the *spreading* traumatic gangrene was at an end. And I will ask you to bear in mind the following facts about this disease, which you must take on my authority: First, that cases occur sporadically without there being any evidence of infection, and that where one case occurs in a hospital ward, it does not tend to spread to the other patients. The best evidence that I can give you of the non-infective nature of this disease is an incident which came to my knowledge some few years ago, and which I think I have on more than one occasion mentioned to you. A surgeon had occasion to amputate a patient's arm for this form of gangrene. The sponges which were used during the operation, and which had been but carelessly rinsed through afterwards, were, by an unfortunate oversight, used next day in an operation for phimosis, and without the slightest detriment to the patient. The circumcision wound healed readily and showed no tendency to run into a condition of septic gangrene. Surely, if there had been anything infective in the nature of these cases, the infection must have been conveyed by the sponges in this instance.

With regard to the fact that it does not spread from case to case in a hospital ward, you will see that in our patient up-stairs I have taken no precautions. I have not isolated him; I have not even surrounded his bed with screens, and the other patients have been allowed free intercourse with him; and this because I recognize the fact that this disease is not of an infectious or contagious nature, and that no harm is likely to accrue to other patients from associating or coming into contact with him.

Now, these five facts which I have mentioned—(1) that the wound was a large, irregular one, difficult to cleanse of all putrescible animal matter; (2) that the spreading of the gangrene ceased after the removal of the limb; (3) that it is a sporadic disease; (4) that there is no evidence of contagion; and (5) that it does not spread from patient to patient, like erysipelas, hospital gangrene, and other allied diseases—seem to me to have an important bearing on the etiology and pathology of the disease.

You know that experiments on animals have shown that there are two distinct forms of disease which may arise from the entrance of putrid animal matter or its products into the blood-stream: one, an acute general affection, not infective in character, which does not increase in the system, in which the symptoms supervene immediately after the introduction of the poison, and in which the effects produced are proportional to the dose; and to this we give the name of “sapræmia,” or “septic intoxication.” The other, also an acute general affection, but infective in character, due to a specific virus, which increases or multiplies in the system, in which there is a period of incubation after the inoculation of the poison before the characteristic symptoms declare themselves, and in which the effects may be produced by the most infinitesimal dose; and to this we give the name of “septicæmia,” or “septic infection.”

In the first variety, *sapræmia*, it has been shown that the blood of an animal after it has been killed by this poison contains no recognizable microscopic organisms, and the poison is therefore believed to be due to some chemical substance, generated, indeed, by organisms, but itself containing none. And, indeed, this poison has been obtained experimentally, and thus obtained may be dissolved in water, forming a clear solution which contains no micro-organisms, but which possesses all the properties of a septic poison, and which, when injected into animals, produces all the symptoms of *sapræmia*.

In the other variety, *septicæmia*, on the other hand, the conditions are very different. Upon examining the blood of an animal which

has died from its effects, it is found to contain vast numbers of micro-organisms, which have penetrated, in some cases, even into the substance of the white corpuscles. If a tiny drop of this blood is introduced into a mere scratch, it may produce similar symptoms in another animal.

I think we must admit that the disease we are considering does not belong to this class. We have seen that it is not infective, and that even the inoculation of some of the blood of a patient affected with this disease into the wound of another patient does not produce any evil results. There are no micro-organisms to be found in the blood of patients who are suffering or have died of this disease, and indeed it would be impossible to understand how, if there were organisms in the blood, the removal of the diseased parts would arrest the progress of the disease. There are, however, innumerable organisms to be found in the affected tissues. I do not see, therefore, how we can regard this disease as a true infective process. It seems to me that we should regard it as more allied to the first class of cases, those of *sapremia*, and should be justified in saying that the general condition of the patient is due to poisoning by the chemical products of putrefaction.

The sequence of events I should explain somewhat in the following way. We get a large lacerated, irregular wound, accompanied often by injury to a bone or joint, which it is exceedingly difficult to free of all blood-clot or serum, and hence these become pent up in the wound. Into this putrescible matter bacteria find their way, and the blood and serum undergo putrefaction. The result of this is the formation of some chemical substance, the product of putrefaction, which infects the tissues locally and sets up gangrene wherever it permeates, and, no doubt, is also absorbed into the blood and produces the constitutional symptoms which I shall detail to you directly. If this hypothesis is correct, we can easily understand how the patient recovers after amputation. The poison ceases to be generated, and if he can only throw off the poison he has already absorbed he will recover. This is very different from a true infective process, where the poison goes on multiplying in the blood, and continues to increase until it has destroyed the patient. That such a process as that I have just suggested does sometimes take place is evident from what is known of the pathology of tetanus.

This disease is believed to be due to the inoculation of a special bacillus whose habitat is earth or mould, which when inoculated into a wound multiplies there, but does not find its way into the general

blood-stream, but remains in the neighborhood of the wound and generates there a poison which is known under the name of tetano-toxin, which is absorbed into the blood and, circulating there, by its action on the nerve-centres, produces the tonic contractions of the muscles and other symptoms characteristic of the disease, and eventually, in the majority of cases, kills the patient. In connection with this point an interesting case, and one bearing very strongly on the line of reasoning I have endeavored to lay before you, occurred in this hospital about three years ago. A patient was admitted under the care of Mr. Rouse, who subsequently developed this form of gangrene of which I am speaking, diffuse or spreading traumatic gangrene. The patient died, and Dr. Slater, when he subsequently examined the gangrenous tissues, found them teeming with this peculiar organism, the earth bacillus, which is characterized by its large size, and is always seen in a state of sporulation, when it resembles a drumstick ; and he was unable to distinguish it from the tetanus bacillus. So that it would appear that there are micro-organisms indistinguishable from each other, found in the earth, which, when admitted into the body, multiply and generate a poison, which, when absorbed into the blood-stream, produces the most deadly effects by acting on the nervous centres and inducing tetanus, and at other times acts locally on the tissues and causes the most virulent and rapidly-spreading form of gangrene. Whether there are two bacilli, closely resembling each other, which produce these different effects, as would seem the more probable, or whether there is only one, which under varying circumstances produces the two conditions, is not known.

I think a further piece of evidence that this form of gangrene is produced by some chemical poison which is generated at the seat of the wound, and not by a micro-organism in the tissues themselves, is evidenced by the case up-stairs. You will remember that in performing the amputation I actually cut through infiltrated tissues, for reasons which I will mention presently, and that, in my posterior flap especially, the subcutaneous tissue was infiltrated with a greenish, semi-gelatinous fluid. It is true that this portion of the flap sloughed, as I expected it would, but there has not been the slightest spread of the disease. Now, if there had been any micro-organisms in this tissue, they would assuredly have gone on multiplying, and the disease would have spread. It would seem, therefore, that these microbes, whatever they may be, can live and multiply only in dead tissue, and a part has to be killed first by the chemical poison which they generate before they can invade and grow in it.

We pass on now to say a word or two on the morbid anatomy of this disease. The cellular planes are the parts principally affected, at all events, in the earlier stages of the disease. If an incision is made vertically through the whole length of the diseased tissues, it will be found that in those parts most remote from the original injury the subcutaneous tissue presents a greenish, gelatinous appearance, being infiltrated with a fluid of this color; and even above this there may be a brownish discoloration, due to pigment formed by the breaking down of blood-corpuscles in the gangrenous area which has diffused itself beyond the seat of the disease. In this infiltrated tissue no micro-organisms will be found. This condition will be noticed to have extended itself further along one side of the limb, generally the inner, than the other. Below this—that is to say, nearer the seat of the original injury—the tissues will be found to be in a state of hopeless gangrene; the subcutaneous tissue will present a sponge-like appearance due to the development in it of gases, the products of decomposition, and to emit a horribly fetid odor. The skin will be involved and the muscular tissue infiltrated. In these parts rod-shaped organisms will be found in great abundance, not only in the tissues, but also in the fluids of the part in which the gangrenous process is fully established.

There are two peculiar characteristics about this particular form of spreading gangrene which deserve especial mention. The first of these is that putrefaction is almost contemporaneous with death. No sooner has the part fallen into a condition of gangrene than decomposition commences, and is attended with the evolution of fetid gases, which permeate the tissues and render them sponge-like, and produces an emphysematous crackling, which is, as I shall have occasion to tell you, an early symptom of the disease.

It also causes an exceedingly offensive odor, which some of you may remember was very marked in our patient up-stairs. The other characteristic point is the fact that the gangrene never stops; there is no formation of a line of demarcation denoting an arrest of the gangrenous process, which in some other forms of mortification is confidently waited for by the surgeon before he institutes any operative interference. To do this in such cases as these would be fatal, for you may be assured that if the process has once started, it will go on spreading until it reaches the trunk, and will inevitably kill the patient. The rapidity with which the disease spreads is sometimes appalling. In our case up-stairs, Mr. Wild, the house surgeon, tells us that when he saw the patient on his morning round the gangrenous

process was confined to the tissues below the knee, and you will remember that when we saw him shortly after one o'clock it had already extended ; at all events there was discoloration as high as the groin. I remember on one occasion admitting a patient with a compound fracture of the leg at the junction of the middle and lower thirds. When I saw him one day during my ordinary round at about one o'clock gangrene was just commencing, but was confined to the tissues in the immediate neighborhood of the wound. I at once told him that amputation was the only means of saving his life. But the man would not consent to undergo the operation until he had consulted his wife. She was immediately sent for, and I saw the man again at six o'clock in the evening, when the wife had been consulted and her permission for the operation obtained. Now the infiltration had extended to the groin, and though I amputated in the upper third of the thigh, the tissues cut through were extensively involved and great sloughing followed, so that when the sloughs had separated there was nothing left but the bone, denuded of all its structures except periosteum, in a bag of skin, which happily had escaped. Fortunately, the man eventually recovered, owing mainly to his being able to take plenty of food and stimulant, which is our sheet-anchor in the after-treatment of these cases.

The disease always sets in before suppuration is established ; generally on the second, third, or fourth day after the accident. At first the symptoms are by no means clear, the edges of the wound become swollen and everted and present a waxy appearance, with vesications around their margins. There is a slight discharge of fetid, brownish-colored serum, but no true suppuration. There is very often a severe pain complained of in the part, and the patient's aspect is distressed and anxious, but at this time there is but little constitutional disturbance, and the case may properly be regarded as one of ordinary septic inflammation, resulting from decomposing discharges, which have been pent up in the wound. But the true nature of the affection speedily becomes apparent. Within a very few hours the wounded limb, at the seat of injury, swells, becomes dusky red, and deep-seated burning pain is complained of. The discoloration, which is at first red or brownish in color, soon becomes of a dusky, purplish hue, and speedily passes into black. The swelling, primarily firm and resisting, becomes soft and doughy, and this is accompanied or almost immediately followed by emphysematous crackling, due to the presence of gases which are developed by the decomposition of the tissues attacked by the gangrene, the putrefactive process immediately follow-

ing the death of the tissues. The disease spreads with great rapidity, being preceded by swelling and dusky redness of the skin, which speedily becomes purplish black, and passes into a condition of hopeless gangrene in a few hours.

The swelling and discoloration extends more rapidly up the inner side of the limb than the outer, where it reaches first the axilla or groin, as the case may be. The portion of the limb below the original seat of the injury becomes pale, cold, and œdematous. The constitutional symptoms speedily assume the character of acute septic poisoning. The pulse is rapid and feeble; the skin hot and dry; the tongue furred; the teeth become covered with sordes; the respiration is hurried and shallow; there is low muttering delirium; diarrhœa, with sometimes bloody stools, and the patient sinks into a prostrate and hopeless condition. The temperature is at first high, but sometimes becomes subnormal before death. Cases almost invariably prove fatal in three or four days from the commencement of the disease.

And now as regards the treatment. There can, I think, be no question as to what is the appropriate treatment. It is to remove the limb at once. If the surgeon trusts to constitutional treatment in the hope that a line of demarcation will form, he will assuredly be disappointed and the patient will certainly die. If, on the other hand, he removes the limb, and if the patient can throw off the dose of poison which he has imbibed, he stands a very good chance of recovering, supposing the amputation is done above the level of the tissues which are infiltrated with micro-organisms, for then there will be no fresh generation of the poison. In our text-books on surgery you will find that great stress is laid on the danger there is of the stump becoming affected with the same disease and spreading traumatic gangrene reappearing in this situation. My own experience would lead me not to dread this, only providing that the amputation is performed beyond the area of actual mortification, and I would ask you to differentiate very carefully between the area where infiltration has occurred and that where the part is dead and where putrefaction is going on; here the tissues are teeming with rod-shaped organisms, and if any of this tissue is left behind, a recurrence of the morbid condition will take place in the stump. This, I say, must be carefully distinguished from the area of infiltration, which is the precursor of the gangrenous condition, and in which, if examined microscopically, no micro-organisms are to be found. If these tissues are left they will most probably slough, but it will be a local gangrene, and there will be no spreading. This was very well shown in our patient up-stairs. You will remem-

ber that I actually cut through and left behind some of these infiltrated tissues, and I did this purposely, for a reason which I will mention immediately, and these infiltrated tissues subsequently sloughed, but there was no further spreading of the disease. It is obviously better, therefore, if you can to cut through healthy structures, so as to avoid the risk of this sloughing, but it is not always possible or perhaps advisable to do so. And this leads me to explain to you why I performed the amputation where I did, instead of cutting through healthy and unaffected tissues. In order to do this I should have had to amputate at the hip-joint, and even then would have had barely sufficient flaps to fashion my stump. Now, this is an exceedingly severe operation, and our patient was in a very weak and critical condition, and, as I believed, scarcely in a state to stand the shock incident to an operation of this severity. Whereas amputation in the upper third of the thigh is a very much less dangerous proceeding, and is not attended by nearly the same amount of shock. I felt that if I amputated at the hip-joint the patient would very probably sink under the operation, and that I was selecting the lesser of two evils by removing the limb in the upper third of the thigh, even though I knew that this proceeding would be followed by some subsequent sloughing. And again, there was the consideration that if the patient recovered he would be in a much better position as regards his future welfare after an amputation in the upper third of the thigh than if he had had the limb removed at the hip-joint. In the latter case he is condemned to spend the residue of his life on crutches, for there is no means by which an artificial limb can be made available for purposes of progression. Whereas in amputation in the upper third of the thigh an apparatus can be fitted to the short stump, on which the patient will be able to get about.

And now one word, in conclusion, about the after-treatment. If our views on the pathology of the disease are correct, we must consider our patient after the operation to be suffering from a dose of septic poison which will produce symptoms proportional to the amount of poison absorbed. What this amount is no one can tell. If the dose absorbed is sufficient to destroy life, the patient will assuredly die; if, on the other hand, the dose is smaller in quantity, the patient will recover, if only he can be kept alive long enough to eliminate the poison from his system. You will see, therefore, that your prognosis should be an exceedingly guarded one. And you will see, also, that your line of treatment is quite clear. You must support and husband your patient's strength by every means in your power. The early and

free administration of wine, brandy, and diffusible stimulants is indicated, with such fluid nourishment as the patient can take and digest. You will find it also advisable to give opium in some form or another, as it allays irritability, relieves pain, and induces sleep, and so assists in enabling the patient to "weather the storm." The issue of a case of this sort frequently depends upon the patient's ability to take large quantities of stimulants. Should he be able to do so, all goes well; but should they cause irritability and vomiting, the patient rapidly sinks from exhaustion.

CHOLECYSTOTOMY AND INCISION OF THE COMMON BILE-DUCT; NEPHRO-LITHOTOMY FOR RENAL CALCULI.

CLINICAL LECTURE DELIVERED AT THE WOMAN'S HOSPITAL OF PHILADELPHIA.

BY JOHN B. ROBERTS, A.M., M.D.,

Professor of Surgery in the Woman's Medical College of Pennsylvania.

THIS patient, aged fifty-seven, was admitted with the following personal history: Two months ago, after a severe vomiting spell, her skin very suddenly turned yellow and the conjunctiva became deeply stained. She vomits everything she eats, clam-broth being the only exception. The bowels are constipated and the movements clay-colored. She has lost thirty pounds in about six months. The lungs and heart are normal. The pulse is slow, not especially weak. She suffers, as do many jaundiced patients, with itching of the skin. She passes about sixty-four ounces of urine, which contains much bile, but no albumin or sugar. Examination of the abdomen discloses what I believe to be an enlargement of the liver on the right side, the dulness extending downward to almost on a line with the crest of the ilium. It was at first thought that possibly the liver was floating, but this was not evident on percussion. As medicinal treatment has failed to relieve the jaundice, I shall make an exploratory incision in the anterior abdominal wall and be governed by the conditions found.

An incision about two inches in length has now been made in the median line of the abdomen between the umbilicus and ensiform cartilage. Exploration of the region about the liver shows the gall-bladder to be greatly distended—six inches long—and to contain fluid. The incision, being lengthened about two inches upward and three inches downward, now measures about seven inches and reaches below the umbilicus. The gall-bladder, which is covered with shaggy lymph, is next punctured near the fundus with a trocar, and I have evacuated a quantity of puriform fluid through the canula. The fundus will now be incised, and a large amount of pus, as you see, escapes, making altogether nine ounces. As I had drawn the fundus of the bladder without the abdomen before puncturing it, the pus does not flow into

the peritoneal cavity. With the finger I remove twenty-one gall-stones from the gall-bladder. Possibly some of these came from within the opening of the cystic duct, which seems enlarged. The opening into the gall-bladder is closed temporarily with forceps to prevent the escape of any remaining fluid into the peritoneal cavity. I feel some masses like gall-stones at what I shall probably find to be the common bile-duct. They are irregular and seem to be faceted. As my attempt to crush these masses within the fingers is unsuccessful, a needle shall be used to pierce the wall of the duct and an effort made to split the supposed stones. This also fails, though, I think, a gritty substance was struck. The common duct is now incised for one inch, and a clear white glutinous fluid escapes into the peritoneal cavity. Of this there is from two to three fluidounces. No stones are found. The duct towards the liver is dilated and a probe is easily passed three or four inches. In the other direction the opening is narrow, but a probe can be inserted an inch or two towards the duodenum. The walls of the common duct are much thickened, but no gall-stones are found. One or two large lymphatic glands which I now find are evidently the masses which I supposed to be gall-stones impacted in the duct. One of them I now divide by the knife to show its character. Some of the nodules may have been the irregular surface of the pancreas.

The opening made in the duct is closed with silk sutures and a drainage-tube conducted to the spot. In the former manipulations the anterior layer of the gastro-hepatic omentum has been torn a little. The torn portion is held loosely in place by means of a silk suture. I now flush the cavity of the abdomen with water at about 110° F. The opening in the abdominal cavity is securely closed with nine deep sutures, and a rubber drainage-tube, which goes to the region of the common duct, is held in place between the sutures. The gall-bladder has been raised by the forceps, which for a time closed the incision in its fundus, and the edges of the abdominal opening near the lower angle are stitched to its thickened walls, leaving the opening to discharge externally. A rubber drainage-tube is inserted into the gall-bladder. All sutures used are silk. The wound is next dusted with boracic acid and the two rubber drainage-tubes are covered with separate pieces of rubber dam. The dressing for the wound is of dry bichloride gauze, held in place by a flannel bandage and binder.

[The tubes were drained with a hard rubber syringe every half-hour at first, the interval being prolonged to an hour as the oozing decreased. The drain-fluid from the upper tube, which was in the gall-bladder, was

yellow and turbid, while from the lower tube a pinkish clear fluid was obtained. Two days later there was no fluid coming from the upper tube, and it was removed.

The patient's condition was quite fair up to the evening of the second day, when she became much prostrated, her pulse grew rapid and feeble, the skin clammy and cold, and she sank rapidly, dying early on the third day after operation. She was treated with quinine suppositories, whiskey, digitalis, and strychnine during the entire after-treatment. A little tympany developed on the second day, for which Rochelle salt was ordered. She urinated spontaneously at various times. A small movement of the bowels followed the use of the laxative.

No autopsy was permitted. An exploration, however, was made of the abdominal cavity through the site of the original wound. Union by first intention was found to have taken place throughout the extent of the wound, and the peritoneal surfaces were united around the gall-bladder. Upon opening the abdomen it was found that there was some free liquid deeply stained with bile; a few adhesions were found between the right lobe of the liver and parietal peritoneum. There had been no bleeding whatever. The intestines were not distended,—in fact were quite flaccid,—and, though somewhat injected, their surfaces were not roughened or adherent. The stomach was greatly distended, its lower border being on a line with the crest of the ilium. The pyloric orifice of the stomach seemed thickened and indurated, while the glands at this site were similarly affected. The aggregate bulk of thickened tissue was about the size of a small fist, and proved to have been the cause of obstruction to the flow of bile. The mass, being cut into, presented the macroscopic appearances of malignant disease. A portion was removed for microscopic examination. The liver, which was greatly enlarged, was severed from its attachments and removed. The sutures in the common bile-duct seemed to have been efficient and union seemed to have taken place.

The immediate cause of death was apparently cardiac failure. It is possible that the exceedingly distended condition of the stomach may have aided in causing death by its pressure upward upon the diaphragm, interfering with the action of the heart and lungs.]

NEPHRO-LITHOTOMY FOR RENAL CALCULI.

This woman, who is of middle age, noticed four years ago that there was a swelling or lump in the right side of the abdomen, but did not observe that it increased much in size until last winter. She then became severely ill, suffered from pain of a lancinating character in the

abdomen, especially when she moved, became much reduced in flesh, and was obliged to remain in bed under medical treatment for eight weeks. Three years ago she had pain in the bladder, and the urine, which was dark, as though mixed with blood, contained a greenish-white sediment. She was troubled at the same time with frequent desire to urinate.

In this case, as in many instances, when we come to investigate the clinical history of a patient, the patient's memory of the sequence of events is untrustworthy. It seems pretty clear, however, that this woman had trouble in the region of the kidney before the symptoms of cystitis arose. Upon examination, I discover that there is a mass within the abdomen extending from the crest of the ilium on the right side upward to the ribs, and having its lower border, on the median side, about three inches below the navel. Upon palpation there is a sensation, such as is given by a hard resisting tumor, which is rather irregular along the upper border. Four small nodules seem to be situated superficially to the larger mass and can be moved by my hand. The mass extends backward into the loin, occupying the normal site of the kidney. A tympanitic note is given by light percussion over the front of the tumor, but deep percussion shows an absence of resonance. This indicates that the tumor is posterior to the intestines. Pressure with the hand placed in the lumbar region lifts the whole mass and gives the patient pain, which is felt especially along the line of the ureter. Slight upward and forward motion is imparted to the tumor by this pressure. The woman's girth is increased by the growth, which is very prominent even on inspection.

Examination of the lungs and heart shows nothing special, except that the heart's action is a little irregular. The urine is alkaline, has a specific gravity of 1020, and is full of pus. A specimen of urine carefully drawn from the bladder so that no vaginal secretion could enter it was found to be half pus. No tube casts were present.

The history of cystitis, and the location of this tumor, which is evidently associated with the kidney, makes me believe that the case is one of pyonephrosis. This may be due to renal calculi lying in the pelvis of the kidney, or to a tubercular or pyogenic infection extending upward from the bladder through the ureter. If the history which the woman gives is correct,—that the pain and swelling in the kidney region preceded the symptoms of cystitis,—it is more probable that calculous pyelitis was the primary condition, and that the cystic symptoms are secondary. If the frequent micturition and symptoms of cystitis came first, I should be inclined to believe that the kidney

became infected by the route of the ureter from the original inflammation of the mucous membrane of the bladder. Examination of the bladder with this steel sound fails to give me the click which is indicative of a calculus within the bladder. Vaginal examination made by Dr. Fullerton seems to indicate that the right ureter is thickened. The patient has no special constitutional symptoms, except debility, and there is no fever. It is very evident that the tumor, which is painful and increasing in size, is only to be treated by operation. The question to be determined is, whether I shall make an anterior incision or a lumbar one. If I were certain that I would extirpate the kidney I should be inclined to make an incision in the anterior abdominal wall, because the bulk of the tumor is such that its extirpation would require a larger opening than it is likely I could make with propriety in the loin. As I believe the case to be one of abscess in the pelvis of the kidney, which has caused great distention, and probable destruction of the kidney structure, I shall make a lumbar incision with the hope that it may be possible to evacuate the purulent or tubercular fluid and remove any calculi that may be found present. A large drainage-tube will then be inserted, and the sac formed from the disorganized kidney washed out daily. At a later period I can, if the case demands it, extirpate the organ by the lumbar route, when the organ has shrunk and the patient has recovered from the debility due to the long-standing disease. This course I think will be safer than making an attempt to extirpate the organ by an anterior incision at the present time. If it were probable that the growth was a solid growth of the kidney, which would then almost certainly be malignant, I should perform anterior nephrectomy.

After proper preparation and etherization an incision three and a half inches long is made in the loin parallel to the last rib, and about midway between the twelfth rib and the crest of the ilium. After division of the muscles and fasciæ, I come upon a pearly-white layer of tissue, which appears to form the covering of the tumor. An incision about half an inch long is made in this tissue, opening up the cavity of an abscess, from which are discharged forty-eight ounces of fetid pus of a greenish color. A finger carried through the opening discovers an irregular cavity within the kidney and a stone in the bottom of one of the pockets. As this stone cannot be withdrawn, a tenotome is used to divide the kidney tissue which encloses it. An irregular triangular stone is now extracted, which from its shape has evidently occupied the pelvis of the kidney. It is one end of this triangular stone which was felt by my finger. Four or five small

stones resembling various sizes of shot are discovered when the cavity is washed with sublimate solution (1 to 1000). These stones I remove with forceps. One small granular one cannot be extracted with the forceps, and in order not to prolong the operation, it is allowed to remain with the expectation of its being washed out with subsequent irrigations.

A half-inch drainage-tube is introduced through the wound into the abscess cavity. The upper angle of the wound is sutured and the whole field of the operation dressed with boracic acid and dry sublimate dressings. These are held in place by an abdominal bandage.

[The temperature during convalescence was afebrile after a slight rise to 100.2° F. the day subsequent to operation. This was the post-operative rise often seen, and which is probably due to absorption of fibrine ferment in aseptic wounds.

After eight weeks' treatment the woman returned to her country home, having been an out-patient for about two weeks. The drainage-tube had been removed, and a small sinus remained, which was being cleansed daily with peroxide of hydrogen solution. Subsequently the woman reported in perfect health with the sinus closed.]

GASTRO-ENTEROSTOMY.

CLINICAL LECTURE DELIVERED AT THE YORKSHIRE COLLEGE.

BY A. W. MAYO ROBSON, F.R.C.S.,

Professor of Surgery in the Victoria University; Honorable Surgeon Leeds General Infirmary and Honorable Consulting Surgeon to the Keighley Hospital.
Member of Council of the R.C.S. England.

GENTLEMEN,—In the first bed in the right of No. 8 Ward is a woman, aged forty, on whom I performed gastro-enterostomy a month ago, and as she is now convalescent and able to take ordinary meals, I will show her to you this afternoon before she returns home. She is suffering from carcinoma of the pylorus, which was diagnosed by my colleague Dr. Chadwick, who sent her to me for operative interference. On admission she looked ill and emaciated, and gave a history of pain for seven months and of persistent vomiting for four months.

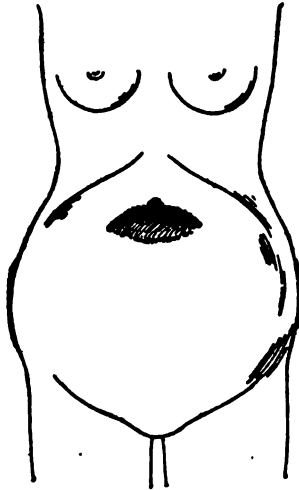
On examination, a hard nodulated tumor, movable from side to side, and moving with respiration, could be felt in the epigastrium, and below this a splashing sound could be elicited on succussion of the abdomen. (See Fig. 1.) On distending the stomach with gas by giving an effervescing draught in separate portions, it was found that the site of the splashing sound was in a dilated stomach. There were no other signs of organic disease. The patient was vomiting daily, and the vomit, which consisted of mucus and undigested food, contained no free HCl, which, though not absolutely pathognomonic, is decidedly in favor of the disease being malignant. The stomach was washed out daily with a solution of boroglyceride, and the patient was fed on peptonized foods, with the result that the vomiting was relieved and she gained over a pound in weight during the first week; but as the improvement did not continue, and as nearly two pounds in weight were lost in the second week, an operation was decided on.

On account of the size of the tumor it was manifestly impossible that pylorotomy or partial gastrectomy could be performed with a view to giving the patient a chance of permanent cure; but, fortunately, by operative interference the food can be made to pass from the stomach

into the small intestine, thus avoiding the obstruction. This may be done in several ways. You have seen me perform the operation (1) by simple suture, without any other artificial help, (2) by means of Senn's bone plates, and (3) in my later cases by the decalcified bone bobbin; and as the last method is at the same time, to my mind, the most simple as well as the most expeditious, and as I have found it by ample experience to be thoroughly safe and efficient, I now give it the preference.

In this case, after the stomach had been washed out with a solution of boracic acid, the lower bowel had been cleared by an enema, and the skin of the abdomen had been aseptitized, the peritoneal cavity was

FIG. 1.



opened by an incision four inches long in the course of the linea alba, the umbilicus forming the centre of the wound. The tumor was found to involve not only the pylorus, but the whole of the lesser curvature of the stomach. The body of the stomach being dilated and free from disease was with the omentum drawn over to the right, and on passing the right index and middle fingers to the left of the spine the jejunum was at once felt and traced from its point of attachment downward, until a free loop of bowel could be brought forward into the wound; this was at once stripped of its contents by holding the extremity of the loop between the right finger and thumb and passing the loop between the index and middle fingers of the left hand, when Mr. Thompson, who was assisting me, secured the base by means of an

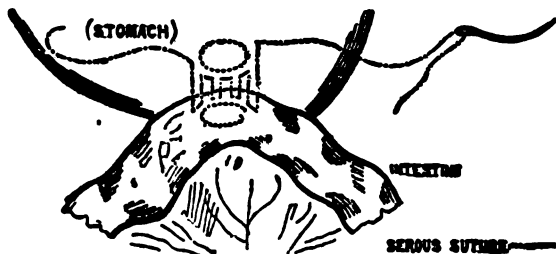
elastic tourniquet composed of a piece of non-perforated elastic drainage-tube, which was tied in one knot and fixed beyond by means of pressure forceps; the left extremity of the anterior stomach wall at its lowest part was then drawn into the wound, and a piece of aseptic gauze moistened with carbolic lotion was placed behind the two portions of viscera exposed, thus temporarily closing the general cavity of the peritoneum. A scratch with the scalpel was then made at the fundus of the intestinal loop on the side turned towards the stomach, and another on the exposed stomach wall, so as to indicate the site of the future openings, which should be about an inch in length. A curved sewing-needle of the size and shape here shown (Fig. 2), pre-

FIG. 2.



viously threaded with fine aseptic silk, was then entered on the left side of the stomach wall, half an inch from the end of the proposed opening, taking up serous and outer muscular coat only; it was then made to take up a similar part of the intestinal surface, also one-half inch from the proposed incision, and so on, alternately stomach and bowel, until the right extremity of the half-circle was reached; the needle was then laid aside but not unthreaded, the parts thus presenting the appearance shown in Fig. 3. By means of scissors the two viscera were

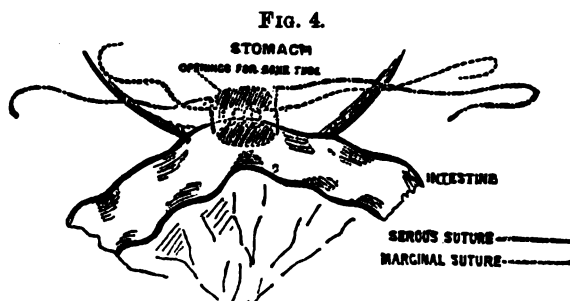
FIG. 3.



Showing serous suture applied around the posterior half-circle.

then opened along the lines previously indicated, and immediately another curved needle threaded with fine chromicized catgut took up the margins of the two openings from left to right, the needle being laid aside but not unthreaded when the right end of the half-circle was completed, as in Fig. 4. The bobbin-shaped tube (Figs. 5, 6, 7) of

decalcified bone was then inserted and held in position by Mr. Thompson until the marginal catgut suture was passed round the anterior half-



Showing the marginal suture applied around the posterior half-circle.

circle (Fig. 8) until it reached the part where the catgut suture commenced, and where the loose end of the suture was found, the two ends were then drawn tight, tied and cut off, thus bringing the margins of the openings, and therefore the mucous membranes of the stomach and

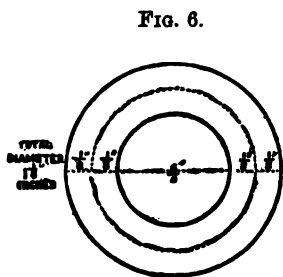
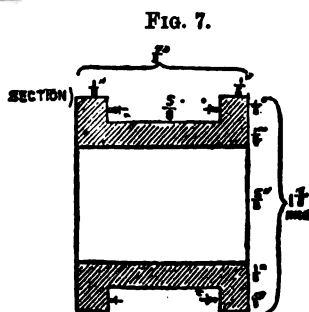


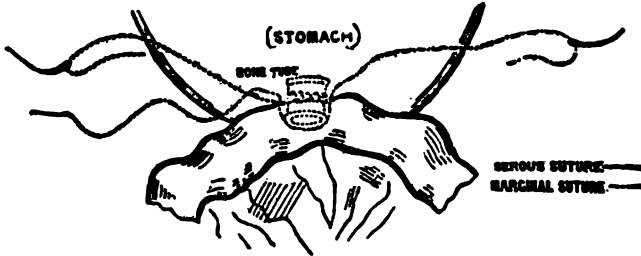
Diagram to show shape and measurements of tube.



bowel, into contact all around; it being impossible to limit the opening beyond the diameter of the bobbin, which at the same time secured immediate patency of the passage between the two viscera and protected the line of suture from the visceral contents. The visceral open-

ings being then closed, the parts were gently bathed with a one in forty carbolic solution, and the anterior half-circle of the serous suture was

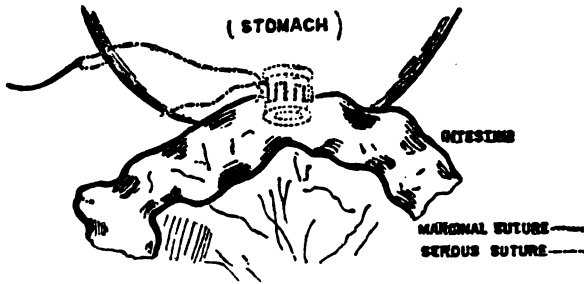
FIG. 8.



Showing tube in position and the anterior part of the marginal suture continued around the circle.

concluded in the same way. (Fig. 9.) When it had been drawn up and tied the suture was completely hidden, and the new fistula was protected by two continuous stitches (Fig. 10), only two needles and two sutures having been employed, and no needle-holder or other appliance having been required. The gauze was then removed, the tourniquet taken off the bowel, the adjoining parts wiped with a sponge, and the stomach and bowel returned, the parts lying snugly on the left side without any tension or apparent disturbance of the normal relations.

FIG. 9.

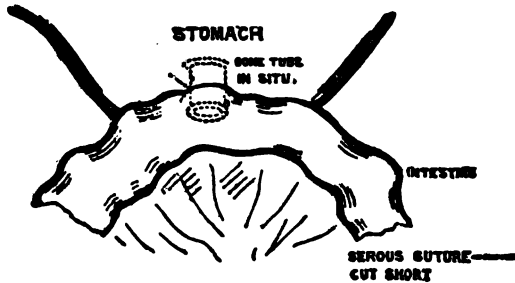


Showing the marginal suture tied and the anterior half-circle of the serous suture completed.

In the after-treatment, nutrient enemata were given for twenty-four hours and sips of barley-water by the mouth; on the second day milk and soda-water and a little tea could be taken, on the third day milk pudding, and on the fourth fish, after which solid food was taken. Beyond a little ether sickness for a few hours there was nothing to chronicle in the after-progress of the case; the wound healed by first intention, and the patient now expresses herself as very comfortable, and as enjoying her food as well as she ever did. She has already

gained several pounds in weight, and curiously enough the tumor is less and seems to be diminishing. In consequence of the irremovable malignant disease relief can, however, only be temporary, and taking the average prolongation of life, she would be likely to survive five or six months; but as her condition is now so good, we may reasonably hope for a much longer relief, as some cases have gone on comfortably for about a year or perhaps more. In a paper published in the Transactions of the Medico-Chirurgical Society for 1892 I pointed out that in two cases in which I had performed the operation by means of simple suture and by the employment of Senn's plates there had been regurgitation of the intestinal contents into the stomach and fecal poisoning. Mr. Barker also had observed the same unpleasant complication. This did not occur in the case I have shown you, and I

FIG. 10.



Showing the bone tube *in situ* and the serous suture completed, the ends being left long to show where the knot has been tied.

think that the complication may in future be avoided by making, as was done in this case, the stomach opening well towards the left, and as low as possible, and by securing a loose loop of jejunum, so that when the union is effected the two apposed viscera will lie snugly and without any tension on the left of the abdomen.

Prof. Braun in order to overcome this regurgitation has performed entero-anastomosis at the same time as gastro-enterostomy, connecting the two limbs of the loop of intestine that has been fixed to the stomach. I need scarcely say that this prolongation of the operation is undesirable if the simpler plan will succeed.

Mr. Jessett's method of making the anastomosis on the posterior wall of the stomach, and Mr. Paul's modification of this by fixing the apposed viscera by means of a ring, offer alternative methods, but I think it will be found that any operative procedure on the posterior gastric surface is attended with technical difficulties leading to an unnecessary waste of time.

The latest modification by Postiknow delays the opening between the viscera until the third or fourth day by avoiding incising the mucous membrane and trusting to a silk suture, tightly applied, cutting its way through. As this method is not only uncertain, but delays the time of feeding by the mouth, which is an important matter in a starving patient, I think it will rarely be practised, as, although the author thinks it safer, we know by experience that there is, with proper precautions, little or no danger of peritonitis, and by the bobbin method absolutely no danger of leakage.

You have seen me or certain of my colleagues perform anastomosis by means of the bone bobbins in quite a number of different cases, such as pylorotomy, enterectomy, ileo-colostomy, and cholecystenterostomy, and the success of the operations in which it has been employed proves that the method can be relied on in practice.

The advantages which I claim for anastomosis by the bone bobbin are,—

1st. Expedition, and this is no small matter in a class of operations where time is an extremely important element. I may mention that I have performed anastomosis by the bone bobbin in fifteen minutes, both in gastro-enterostomy and in ileo-colostomy.

2d. Continuity of mucous surfaces through the new channel, thus avoiding the formation of cicatricial tissue in the healing process, and therefore a limitation of the fistula by subsequent contraction.

3d. Absolute security against leakage by the employment of a double continuous suture.

4th. The certainty of having an adequate and immediately patent opening between the anastomosed viscera.

5th. Simplicity and ease of performance, only two sutures being employed.

6th. The universal application of the method to

- (a) Lateral intestinal anastomosis.
- (b) Lateral implantation as in ileo-colostomy.
- (c) Gastro-enterostomy.
- (d) Pylorotomy.
- (e) Enterectomy.
- (f) Cholecystenterostomy.

The tubes that are being handed round you will see are in three sizes, the largest being specially for large intestine and the smallest for cholecystenterostomy; the one I employed in the subject of our lecture being of medium size, the measurement being given in the diagrams.

HIP-JOINT DISEASE CONTRASTED WITH FLAT-FOOT AND EPIPHYSEAL SEPARATION OF THE FEMUR; THE TREATMENT OF COXALGIA.

CLINICAL LECTURE DELIVERED AT THE BELLEVUE HOSPITAL MEDICAL COLLEGE.

BY REGINALD H. SAYRE, M.D.,

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GENTLEMEN,—Here is a little girl who first came to me two weeks ago, with a history of having had a limp for only about four weeks. You notice when I place her before you that she stands on her left leg with the right foot turned outward; that the right knee is flexed, and that she bears her weight entirely on the left leg. (See Figs. 1 and 2.) The gluteo-femoral crease on the right side is not well marked, and the right buttock is flattened. Please note that she says she has pain in her right *knee*. For the past two weeks she has been lying in bed, and for that reason she walks much better than she did when I first saw her. Now, when I lay her down on her back on this table and examine the various motions of the left lower extremity, I find they are all normal; but when I attempt to rotate the right limb a little, resistance is immediately felt, and I cannot flex the thigh on the abdomen to the full extent. When I abduct the leg, there is more or less spasm of the adductor muscles; the contrast between this and the left side is very marked. You will also notice that when both legs are brought down on the table the lumbar spine is arched forward, showing contraction of the psoas and iliacus muscles. Now, when the left limb alone is on the table, while the right is elevated, there is no such arching; it only occurs when the right limb is placed flat on the table. This arching of the spine may be due to perityphlitic abscess, to psoas abscess, to contraction of the psoas muscle from disease of the spine, or to disease of the right hip-joint. If from disease of the spine, the various motions of the hip would not be impaired as they are in this case. The same would be true if there were a perityphlitic abscess, and we will therefore exclude both; besides, we would probably be



FIG. 1.



FIG. 2.



FIG. 3.



FIG. 4.

able to detect a fulness in the right iliac fossa in either of these cases, which we cannot do here. As this child walks, you observe that the right leg seems to be much longer than the left; but when I measure the distance from each anterior superior spine of the ilium to the internal malleolus of the corresponding leg, I find the legs of equal length. The apparent elongation is due to abduction of the limb, and I would here call your attention to the necessity of placing both limbs in a similar relation to the trunk when taking measurements for comparison, as the distance from the anterior superior spine of the ilium to the malleolus is greater when the limb is abducted than while adducted, and greater when the limb is in complete extension than when flexed.

When a child like this is brought to you for examination, with a history of a slight limp and some aches and pains, do not think the child has rheumatism, or "growing pains," or something of that kind, but make it a rule to strip these children in every instance and examine them systematically, as I have done. I have caused her no pain so far, because I have been careful not to exceed certain limits in making the motions. Now, if I press behind on the right trochanter, or if I flex the limb behind a certain point, she immediately cries. The limitation of motion which you observe here is due to reflex muscular spasm, and is nature's method of guarding a diseased joint.

KYLLOSIS.

Now, contrast the gait of the last child with this next one. She has paresis of the lower extremities and marked flat-foot, and has been brought because she limped and complained of pain. I present the case to you in order that you may familiarize yourselves with the different kinds of limp. Notwithstanding her tender age and her light weight, she has already begun to have pain in the feet. It does not follow that because a child has flat-foot, it may not have hip-disease at the same time; so we shall lay her down on the table and go through an examination of the motions of the joints as in the other case. You observe that they are entirely normal. The limp in this case is due to muscular weakness and flat-foot; in the first child to an inflamed hip-joint.

DIASTASIS OF THE LEFT FEMUR.

Here is a third child. From her manner of walking, you might think at first that she had hip-disease. The right limb appears to be longer than the left, but you notice that I can turn the right foot inward, which I could not do in the first case. On laying her down, you see the difference in the length of the limbs is real and not ap-

parent, as in the first child, the right leg being the longer. The motions of both hips are not at all restricted, so we can exclude disease of the hip. Taking a string, and passing it from the anterior superior spine of the ilium to the tuberosity of the ischium, I have what is called Nélaton's line. Under normal conditions, this line should pass across the tip of the great trochanter. In this child the trochanter on the left side is one inch and a quarter above this line; the right trochanter is in its proper position. Her gait is in reality due to the shortness of the left leg. The child has had at some time a diastasis, that is, a separation of the shaft from the head of the bone, or else a fracture of the neck of the femur. Just when this occurred I have been unable to ascertain. There is no limitation of movement, no pain, and no tenderness. All this child needs is a high shoe to enable her to walk properly, and, as you see, when this is applied the limp disappears.

Now, to return to the first case. When I press against the feet, the child complains of pain; but she says the pain is in the knee. This is because the obturator nerve which supplies the hip-joint also supplies the knee, and hence the pain is referred to the knee when the hip-joint is diseased. It is just the same when a child complains of pain in his hip or thigh, and the disease is in the spine; or when one has pain in the fingers from a blow on the "funny-bone." The pain is referred to the distal extremity of the nerve, and not to that part of it which is in proximity to the inflamed area, and I must caution you against being misled by these sensations of pain, and so searching for disease where it does not exist. What is to be done with this little girl? The child may, perhaps, not recover with perfect motion of the hip-joint, but it should, at least, recover with the limbs parallel; and the first step towards this result is to give this inflamed joint rest, and thus put a stop to the muscular spasm that is causing the deformity.

Now, having placed this child flat on her back, and got the anterior superior spines of the ilia on a line at right angle to the median line of the body, we observe the relation of the diseased limb to this line and to the horizontal plane. The right lower extremity is elevated at an angle of forty degrees, abducted about ten degrees, and the toes are turned out, and the leg cannot be lowered or brought more nearly parallel with its fellow without arching the back or twisting the pelvis out of its present relation to the trunk. The position which the diseased leg assumes when the back is flat and the two anterior superior iliac spines are on a line at right angles to one passing from the sternum to the symphysis pubis, is called "the direction of the deformity." (See Figs. 3 and 4.) You must not apply a hip splint with the limb in this

distorted position ; you must first reduce the deformity. To do this, place the child in bed with a well-padded, long, straight board splint, extending from the axilla to the foot of the sound side, and with a piece of board at right angle to this below the feet to keep the splint from tipping. The child must be securely bandaged to this splint in order to keep the body still. Then apply adhesive straps to the affected limb, and then make extension in a line with the deformity, by means of a weight fastened to the plasters by a cord running over a movable pulley, gradually lowering the limb as the spasm of the muscles about the hip relaxes under the influence of rest, until it can be placed on the bed without any tilting of the pelvis. Not until then must you attempt to apply a straight hip splint. The adhesive plasters should always be made to extend above the knee. If this is not done, after extension has been carried on for some time there will be a relaxation of the ligaments of the knee, and disease of the knee-joint may be excited simply as a result of the extension. Having laid the plaster strips, which should be of heavy moleskin extension plaster, one on either side of the limb, I apply a roller bandage over them very tightly for a few minutes, until the plaster has become adherent to the skin ; then I remove this tight bandage, and apply one less tightly from the toes up to the upper part of the thigh. A weight will be attached to the buckles at the lower ends of these adhesive straps by means of a wooden "whiffle-tree," which fastens by leathers to the buckles, and the limb will be placed on a pillow so as to secure traction in the line of the deformity.

When the disease is more advanced than in this little child, you will find that the slightest motion causes pain ; there is present a very marked muscular spasm ; and when the child goes to sleep the muscles yield a trifle, causing a slight motion of the joint and severe pain ; and, as a result, the child awakens with a shriek. You will find in such cases that traction made in the line of the deformity will cause relaxation of the muscular spasm, while traction improperly applied causes pain by increasing the tension in the hip-joint. If there be much effusion into the joint, a blister applied behind the great trochanter will be found a valuable means of aiding its absorption, and in exceptional instances it will be necessary to resort to aspiration to remove the fluid.

Here is a little boy whom I saw two years ago, when he had much the same symptoms as you have seen in the first case to-day. Some time before I saw him he began to limp, but the limp improved without any treatment,—as it often will,—and consequently no treatment was instituted until the limp again became worse. When I saw the

child, there was limitation of motion in all directions ; there was flexion to forty degrees, the foot was turned outward and the leg abducted, and there was pain behind the joint on pressure. The disease was in the incipient stage,—the time when we must be able to make the diagnosis in order to secure the very best results from treatment. To-day I find this boy is nearly well, and I shall therefore remove some of the high sole from his shoe so that he can walk on the brace, the brace then serving only to steady the joint.

In certain cases seen at a quite early stage you will get cures with absolutely perfect motion, but you cannot expect such a result in the majority of cases of hip-joint disease.

On examining this boy, I do not find any joint-spasm ; if there should be any spasm of this kind and the splint were removed, the spasm would almost surely increase. All that I find in this boy is a very slight rigidity on the affected side, probably from disuse. I can bring up the limb to a right angle, but not as far as the one on the other side. I think this will improve. Both limbs, however, can be flexed together up well on to his chest ; he can cross the affected limb over the other leg pretty fairly, and I think after a little practice he will be able to do this much better. I consider him perfectly well, but it is hardly safe to remove the splint at once.

This other little fellow, however, is surely out of the woods. He was first shown at this clinic two years and a half ago, at which time he had incipient hip-disease. He wore a long traction hip splint for two years. This was taken off last fall, and as he has returned to-day for observation, I thought it was worth while to show him to you. You will notice that there is a little shortening of his right leg, and this shoe has to be made about one-fourth of an inch thicker in the sole than the other. There is no other abnormality present. He can bring either knee up to the chin, and can cross either leg over the other without the slightest difficulty. (See Figs. 5, 6, and 7.) This is an exceptionally fine result after treatment for hip-disease, and one which should not be held out to the parents when asking a prognosis, as you may be disappointed if you are too sanguine at first.

Here is another patient. You notice that, as this young man stands he favors the left limb, and that there is a slight obliteration of the left gluteo-femoral fold ; but beyond this, inspection reveals nothing. When he lies on his back and the limbs are rotated, there is no resistance to motion of the right lower extremity, but marked resistance to inward rotation of the left limb. The circumference of the right thigh at the middle is forty-four centimetres, and of the left thigh forty-five



FIG. 8.



FIG. 7.



FIG. 6.



FIG. 5.

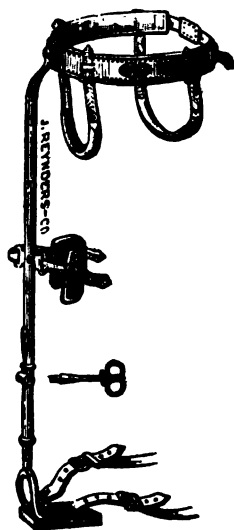
and a half centimetres. Ordinarily, we find atrophy on the diseased side; in this case the increased growth is not muscle, but is due to a brawny deposit deep down in the neighborhood of the inflamed joint. On attempting to flex the limbs the right flexes perfectly well, but the left thigh is stopped at a right angle, and adduction of this thigh is also restricted. When I press between the left trochanter and the anterior superior spine,—in other words, over the head of the bone,—he says I cause him pain, and at this point I can detect deep fluctuation. There is an effusion into the joint, but this cannot be very great, otherwise I would not have been able to move the joint so much. If there were much effusion into the capsule, the limb would be flexed, everted, and abducted, because, in this position, the capsule is the most relaxed and can hold more fluid. When I make traction on the limb, he says it “feels good;” on the other hand, when I push upward and press the joint-surfaces together, as occurs in the act of walking, it causes pain. Now, we have here a plain indication for treatment. We must prevent this traumatism to the diseased joint, and we shall do it by the application of broad strips of Shiver’s yellow plaster to the limb, by which we shall be able to make traction on the limb with the aid of a hip splint. The foot should also be bandaged to prevent it from swelling.

Here is still another boy; one of those cases where there has been a limp for some time, but no pain. Children often say that there is no pain, and yet the child’s face expresses evident pain. Children are apt to deceive you in this respect from fear of your inflicting more pain by manipulation. This boy was brought to me some time ago, and at that time he had been limping six months. He bore the weight of the body on the left leg, and the right leg was flexed; movements of the right leg beyond a certain point caused some pain, but the limb could be moved, nevertheless, through a considerable arc before it hurt him. He was put to bed with a padded board all along the sound side, and traction made on the diseased leg in the line of the deformity.

As he lies on the table now, you see that the back arches a little. (See Fig. 8.) He has been treated at home by extension in the line of the deformity in the manner already described, but as the flexion has not been completely overcome, this treatment in bed must be continued a short time longer. There is often a swelling observed in these cases behind the trochanter, and it means the formation of a deep abscess. These cases almost always begin as an osteitis, and this is frequently associated at first with violent pain. This pain will often subside very suddenly, owing to the escape of pus into the soft tissues and the im-

mediate relief of the tension which has produced the pain. In this boy even slight inversion of the foot causes a tipping of the pelvis, owing to the rigidity produced by the muscular spasm. This is a good example of the way in which this muscular spasm protects the diseased joint. As long as I do not flex the limb very much, he has no pain; but as soon as I pass beyond a certain point, he cries out. I now make slight traction on the leg, and he says it "feels good." This is just what I want, and I shall try to make him "feel good" all the time by the application of this hip splint, which, by means of adhesive plaster and the ratchet movement, secures the desired extension on the limb. (See Fig. 9.) The splint, which I shall apply when his leg is flat, consists of a pelvic band which comes just below the anterior superior spine of the ilium. This is attached by a platform joint to a bar which passes downward on the outer side of the limb to a little below the foot, where it terminates in a foot-piece shod with leather to prevent slipping, and supplied with two straps to attach it to the buckles on the lower ends of the adhesive plasters in the same way as the weight and pulley were fastened. It should be sufficiently strong to support the weight of the body without bending. In order to obtain the maximum of strength with the minimum of weight, the bar is flattened antero-posteriorly. The perineal straps, which are padded, should be attached to the pelvic band near together in front, so as to avoid making pressure on the femoral vessels, and wide apart behind to keep away from the anus. If this be neglected it will cause much discomfort, and perhaps a good deal of œdema of the limb. The shaft of the instrument can be shortened or lengthened by means of this ratchet movement. This splint should be applied while the child is lying down. In many cases they sleep better in this splint, but in others it will be found that the padded board and weight and pulley are more convenient at night. The perineal straps should be covered with linen, which can be easily removed when soiled, and there should be two pairs of these straps, so that one pair can always be clean. When you put on the child's shoe, some one should always hold the limb steady, so that injurious pressure will not be made on the diseased and tender joint. The shoe should also be cut open to the toe, in order that it may slip

FIG. 9.



on easily. The splint must be so adjusted, by means of the perineal straps, that the pelvic band comes below the crest of the ilium, the straps attached to the foot-piece are then buckled to the buckles on the ends of the adhesive straps, and the amount of traction is regulated by means of the key to the point at which the child tells you the traction is comfortable. The foot-piece is sufficiently below the foot to allow the latter to swing clear of it when the patient is walking. The splint acts as a crutch and keeps the weight of the body off the inflamed joint-surfaces, and the slight traction exerted by these straps is, in my opinion, a great advantage over that form of splint which acts simply as a crutch. As the splint projects some distance below the foot, the sole of the shoe on the sound side is elongated by cork or wood to a sufficient amount to make the patient walk easily,—from two and a half to three and a half inches, according to the size of the patient.

In some instances you will find both hips involved, and then the only practicable treatment is recumbency, with traction, in the vast majority of instances; but if the children can be so arranged that it is not necessary to confine them to the house while recumbent, a long step has been made towards building up their general health, and I am happy to be able to show you to-day a case that illustrates this principle most clearly.

This little boy first came to see me about fourteen months ago, because of some difficulty about the knee which had existed for about one year. On examination I found inflammation of both knees, of the right hip, of both ankles, and of the lumbar spine. He was excessively tender, and movement of any of the joints caused exquisite pain. On account of the large number of joints affected, the only practicable way of treating him was by the wire cuirass. It is simply a portable bed which keeps the child quiet and comfortable, and at the same time allows him to be taken into the open air and sunshine. His right hip-joint was flexed to an angle of forty-five degrees, and the right knee to about forty degrees, and markedly subluxated. The limb was, therefore, placed in the cuirass in this position, and so supported by padding placed underneath; while, by the thumb-screws attached below the foot-pieces, traction was made, and counter-traction was secured by padded straps in the perineum, similar to those used on the hip splints and fastened to the cuirass in the same way. I have only seen the child a very few times since then, yet the father has been able to carry out the treatment very well; and although the child was in a wretched condition when first seen, you observe that he is now fat and hearty. The joints of the legs are so much better that the extension plasters are no

longer needed, and have, therefore, been removed. A pad was placed behind the head of the right tibia, and, by bandaging, the sublaxed head of the tibia was forced forward into place. You cannot appreciate, except by personal observation, the great torture these children suffer in advanced cases of joint-disease until their joints are placed at rest. As a result of this suffering they are unable to sleep, their appetite is lost, and their general nutrition becomes seriously impaired.

Now that I have taken him out of the cuirass, you see that the motion of the ankle is quite good. When last seen, he would not allow me to move the left knee-joint at all; now it can be moved somewhat without causing him pain. When he told me it hurt to move it, I did not attempt to do so, and put him back in the cuirass to wait. Whenever you find the muscles rigidly contracted about a diseased joint, do not be in a hurry to move the joint. Nature is doing her best by muscular spasm to prevent any motion, and give the joint a chance to get well, and will succeed if not prevented by over-zealous interference with her methods. In the right knee there has been some involvement of the articular surface, and the shape of the joint somewhat changed in consequence. The motion of the right hip is now excellent, though it was extremely limited when the child was put in the cuirass. Turning the child in the prone position, and passing the finger along the spinous processes, we find one small and almost imperceptible "knuckle" in the dorso-lumbar spine, which represents the seat of the disease. The treatment for these inflamed joints is physiological rest, and nature will repair very extensive damage if you will only give her a chance.

Now, notice the attitude of this little girl. You see that the left limb is flexed, and that the foot is very much more everted than in the children whom I showed you just now. The left gluteal fold is also nearly obliterated. The child suffers pain, which is referred to the left knee. Examination shows the left knee sublaxed backward, the tibia rotated outward, and a puffiness over the inner condyle of the femur, and evident chronic disease of the knee-joint; but further examination shows, in addition to this tubercular disease of the knee, that the right hip is apparently ankylosed so that the body moves with the limb. If, however, I steady the pelvis with one hand, and endeavor to flex the limb while I make slight traction, I find that flexion can be made through a limited arc. If I now bring down both limbs so that the popliteal space on each side comes in contact with the table, I find the lumbar spine is arched up from the table. Examining the motion of the left hip, I find here that the cause for that which takes place when the

limbs are lowered separately, also an attempt at flexion is immediately resisted by muscular spasm, and that the tensor vaginæ femoris can be felt as a rigid band under the skin. The other day when I examined the flexibility of the spine, by placing the child face downward on the table and raising the lower extremities, I found distinct limitation of motion in the spinal column. Since that time the child has been at rest, and this rigidity is not so marked now as then, although distinctly perceptible. We have here disease of the spine, the left knee, and both hips.

I shall now place her in a wire cuirass with the body in such a position that the anus comes over the central opening in the cuirass, thus enabling the child to defecate and urinate without being removed from the apparatus. On the side where there is no disease of the knee, I shall apply adhesive plaster straps on either side of the limb up *above* the knee. On the side where the knee is diseased the plasters extend only to the head of the tibia. You notice that, as the mother put on the stocking, the child cried, because in doing this the limb was jarred, and the tender joint-surfaces were pressed together. This is often one of the first symptoms which attracts the attention of the parents.

In all of these cases which I have shown you to-day there has been pain at the knee, and in some there has been disease in the knee-joint; but in others there has been disease in the hip, and I would again caution you to look for trouble not only at the points where pain is present, but not to forget that the pain may simply point to disease in a remote joint. The rubber adhesive plaster which some use for making traction is very inferior to Shiver's yellow adhesive plaster, for, although very convenient to apply, it is much more irritating to the skin, and consequently cannot be left on for nearly so long a time. I have frequently applied Shiver's plaster, or Maws's English mole-skin plaster, and have been able to leave it on for twelve months. As the thighs are flexed on the body, it is important to place cotton batting under the limb so that the spine may be allowed to rest flat on the cuirass, while the limb assumes its deformed position. You may consider a point like this hardly worthy of your attention, but it is just such little details that make all the difference between success and failure in the treatment of chronic joint-disease. The plasters which I have applied are now fastened around the movable foot-pieces of the cuirass, and a proper amount of traction is made by turning the thumb-screws which move the foot-pieces. If I did not make provision for counter-traction, however, the child would simply slip down in the cuirass; hence, it is necessary to apply a bandage around the handles of the cuirass, passing between the legs and back again to the handles several times, so as to

fix the body, or else to use perineal straps like those employed for the last child. Enough traction is made to make the child comfortable. Little by little, as the treatment proceeds, we shall remove the cotton batting and lower the limb. This child will be kept in this cuirass until the inflammation in the joints has subsided enough to make it wise to remove her, probably two years or so, during which time she can be carried about out-doors without jarring and pain. Care must be taken that she does not become chafed and that the skin is kept sound, but you have seen by this other child's condition that it is perfectly practicable to keep the child free from galls and sores if you are careful.

It may be necessary to add to this cuirass a "jury-mast" or support, passing from behind the head of the apparatus over the child's head. A cross-bar is attached to this by an elastic band, and to this is fastened a leather head-support passing under the chin and occiput to make traction on the spine. As the disease is low down in this child's spine, and it is to be kept horizontal almost all the time, it may, perhaps, be unnecessary to add this head-support, as the disease is still very incipient.

**RETRO-PHARYNGEAL ABSCESS; SARCOMA OF THE
CÆCUM, SIMULATING APPENDICITIS; EXSTRO-
PHY OF THE BLADDER; NECROSIS OF THE TIBIA
FOLLOWING EPIPHYSITIS.**

CLINICAL LECTURE DELIVERED AT THE CHILDREN'S HOSPITAL, PHILADELPHIA.

BY HENRY R. WHARTON, M.D.,

**Demonstrator of Surgery in the University of Pennsylvania; Surgeon to the Chil-
dren's, Presbyterian, and Methodist Episcopal Hospitals, Philadelphia.**

GENTLEMEN,—The first patient I bring before you to-day is this little child, sixteen months of age, who was brought to the hospital two days ago, with the following history :

For about two weeks there was some swelling beneath the angle of the jaw on each side, most marked upon the right side. The child had experienced at times some difficulty in swallowing, and twenty-four hours before admission to the hospital had developed dyspnœa, which was gradually increasing.

When I saw the patient a short time after admission to the hospital, I found her restless, with an anxious expression, marked dyspnœa, and some cyanosis of the lips. Upon holding the jaws open by means of a mouth-gag, I found that the tissues of the pharynx were very much swollen, and upon exploration with my finger I could feel a mass bulging into the pharynx in which I could distinctly feel fluctuation. I considered it a retro-pharyngeal abscess, and decided that it should be opened promptly. However, before attempting to open it, I had the instruments for tracheotomy and a tracheotomy tube at hand in case it should be necessary to resort to this operation, if the dyspnœa became more alarming. I introduced a mouth-gag and separated the jaws, and an assistant held the child's head, and I introduced the blade of a straight bistoury, which had been wrapped with plaster to within half an inch of its point; guiding this with my finger placed upon the swelling, I incised it, and there immediately escaped a quantity of thick purulent matter. The child was quickly turned over on its face, and its body was elevated to facilitate the escape of the pus and to prevent

its being drawn into the larynx. As soon as the abscess was opened the dyspnœa diminished and the child seemed much more comfortable. The after-treatment of the case consisted in spraying the throat with Dobell's solution, and in the administration of quinine and a moderate amount of whiskey.

I bring her before you to-day to make an examination of her throat, and to see if there has been any further accumulation of purulent matter in the abscess-cavity ; and, upon introducing the mouth-gag and passing my finger back into the pharynx, I feel quite marked thickening of the tissues of the posterior wall of the pharynx behind the right tonsil, but can detect no fluctuation in the mass. Her condition is so much improved that she will be able to leave the hospital in a few days.

Retro-pharyngeal abscess is quite a rare affection, and is more apt to be seen in young children than in adults. The purulent matter occupies the connective tissue between the cervical vertebræ and the pharynx and bulges into the pharynx. Retro-pharyngeal abscesses may be met with in tuberculous children, and here often arise from caries of the cervical vertebræ, or may arise from direct infection in the course of scarlet fever or measles, or they may be of glandular origin. The abscess formation is usually slow, and, as the purulent collection increases in size, there may be developed difficulty in deglutition and finally difficulty in respiration. The history of a case of retro-pharyngeal abscess is usually very similar to the history of the case that I show you here to-day ; the disease is extremely obscure at the outset ; the child may be restless, refuse to take nourishment, and exhibit a certain amount of pain in the beginning of the act of deglutition. It is said that painful distortion of the features when drinking, and also that a snoring sound in respiration, especially during sleep, should always arouse your suspicion of this affection. Inspection of the pharynx will often reveal nothing more than thickness and reddening of the pharyngeal mucous membrane, which is coated with mucus. As the abscess increases in size difficulty in swallowing becomes more marked, and finally dyspnœa may be a prominent symptom, which may often be accompanied with cyanosis of the face. Another very prominent symptom is the expression of extreme anxiety which the patient shows. In certain cases there is present, as in the case you saw to-day, some swelling of the neck behind the angles of the jaw. The dyspnœa and cyanosis may be confounded with those conditions which are present in cases of croup, but the slow development of the dyspnœa and the fact that there is not usually cough or suppression of the voice, would enable you to eliminate this disease.

From the symptoms I have given you, of course, you could not make the diagnosis of post-pharyngeal abscess alone, and it is only by exploration of the pharynx with the finger that you can arrive at a positive diagnosis of the affection. Upon exploring the pharynx with the finger, you can usually feel a tumor as large as a walnut bulging into the pharynx, in which it is almost always possible to distinctly feel fluctuation. There is no form of abscess in which a prompt incision is more urgently indicated than in cases of retro-pharyngeal abscess, for death has occurred in these cases from rupture of the abscess and aspiration of its contents into the air-passages. So as soon as you have ascertained the possible presence of pus in a swelling in this locality, it should be promptly incised. The abscess can be opened with a sharp tenotome with a short blade, or you can use a straight bistoury with the blade wrapped with plaster to within half an inch of its point, and, as it is usually impossible to expose the abscess freely, you have to guide the point of the instrument by means of your finger in making the incision. Retro-pharyngeal abscess may also be opened by an incision from the neck, and in that form of retro-pharyngeal abscess which arises from caries of the vertebræ this is probably the best position at which to make the opening, as it permits of good drainage and gives better means of applying local treatment to the abscess-cavity and the sinus resulting.

The prognosis in cases of retro-pharyngeal abscess arising from caries of the cervical vertebræ is not so favorable as in those cases in which it arises from infection in the course of scarlet fever or measles, or is of glandular origin. In the case I have just shown you there is no evidence of spinal caries, nor has the child recently had scarlet fever; and the abscess probably has resulted from a cellulitis of the post-pharyngeal connective tissue, and I am, therefore, inclined to give a favorable prognosis.

SARCOMA OF THE CÆCUM, SIMULATING APPENDICITIS.

The next patient I bring before you is a little girl less than two years of age, who, several months ago, was sent from the surgical dispensary for admission into the house, presenting symptoms which very closely simulated appendicitis. The patient is a Russian Jew, and the history of the case could not be very clearly made out; but from what could be obtained it seems that the patient had been suffering with fever and more or less diarrhoea for several weeks before admission to the hospital, and when I examined her upon admission I found that she was rather emaciated and had a moderate elevation

of temperature; the abdomen was somewhat distended, and a mass could be distinctly felt in the right iliac fossa. As her symptoms were not urgent, she was placed in bed and was carefully watched for several days. During this time it was noticed that the bowels were inclined to be a little loose, that there was no marked distention of the abdomen, and that her fever gradually diminished, although the mass in the iliac fossa increased steadily in size and fluctuation became more distinct. The possibility of the mass being a sarcoma was also considered, but the absolute diagnosis was not positively made. I decided that it would be wise to make an exploratory incision to ascertain the nature of the swelling and drain an abscess if it were found. Upon cutting through the abdominal walls and entering the peritoneal cavity, I exposed a mass resembling granulation tissue covering the cæcum, which bled freely, and there was some escape of bloody serum. The mass was so large and so firmly attached, and the child was so very much shocked, that I deemed it unwise to make any attempt at its removal; having secured some small fragments from the growth, after applying some iodoform gauze packing to control the hemorrhage, I closed the wound with sutures. The child reacted well, and I show the patient to you now, several months after the operation, with the wound perfectly healed. The child is rather anæmic and emaciated, but takes food well, has no obstruction of the bowels, and you can see, on examination of the abdomen, a large mass which projects from the right iliac fossa. A microscopical examination of the fragments from the growth proved it to be a small round-celled sarcoma. I see no indication at present for operation in this case, for, as I previously stated, the child is comfortable, takes food well, has regular movements of the bowels, and I do not think that the patient would stand so severe an operation as the removal of the growth, which is evidently attached to the cæcum, and there have recently developed symptoms of orbital sarcoma, which is an additional contra-indication to operative interference. The most interesting part in the case is the similarity which the case presented to one of appendicitis in its earlier stages.

EXSTROPHY OF THE BLADDER.

This little boy, D. W., three years of age, I bring before you to-day to show a very marked congenital defect of the bladder due to arrested development during foetal life, consisting of an absence of the anterior wall of the bladder, with a corresponding deficiency of the lower part of the abdominal parietes and separation of the pubic symphysis. This

condition is known as exstrophy of the bladder. The deformity is very apt to be associated with a malformation of the penis in the male (which is epispadic), or of the clitoris in the female, which is split in two portions corresponding to the nymphæ. Marked inguinal herniæ are frequently associated with this condition. You see protruding from the lower portion of the abdomen of this patient a purplish-red mass covered with mucous membrane, and at the lower portion of this mass two small openings may be seen, from which urine escapes, which are the orifices of the ureters. The malformed penis is epispadic.

You can see, from the conditions presented, that a patient suffering from this malformation is in a very miserable condition, with absolutely no control over the escape of the urine, which, dribbling constantly over the scrotum, thighs, and back, produces great irritation of the skin, and the clothing is constantly soiled and becomes offensive. The child is now about three years of age, and it is a favorable time to attempt an operation for the relief of this deformity. The operations which are performed for the relief of exstrophy of the bladder have in view simply the formation of a covering over the exposed bladder, and so put the patient in such a position that he can wear an apparatus to collect the urine and prevent its soiling the clothing and irritating the skin. All the operations which have been proposed and practised for the relief of this deformity are attended by a certain amount of shock, due to the loss of blood and to the extensive dissection of flaps which are required to cover over the bladder, so that I do not think it advisable to attempt the operation in very young children or infants; it is better, I think, to wait until the child has reached the age of three or four years before the operation is undertaken.

A number of operations have been devised and practised for the relief of this deformity, and the best of these seems to be that of Mr. Wood, of London, which is the one I will perform in this case, and which consists in making three flaps, one of which is taken from the umbilical region and is inverted over the bladder with its skin surface in contact with the mucous membrane; the others are taken one from each groin and united in the median line over the first flap, with their raw surfaces in contact with the raw surface of the inverted flap. You notice in cutting these flaps I am very careful to control any hemorrhage which occurs, and after the flaps have been dissected loose I secure them in position by means of silkworm-gut and silk sutures. I next tie any vessels which bleed, and then attempt to close the gaps from which the flaps have been taken, as far as possible, by means of sutures. You see I am able to close the gap entirely in the anterior wall of the

abdomen, from which the large saddle-shaped flap was taken, by using heavy wire sutures. This is of decided advantage when it can be done. Having secured the flaps in position, I next apply an antiseptic gauze dressing and have the child placed in bed ; and you notice that, although the operation is a somewhat protracted one, the child is not suffering to any great degree from shock. If the flaps all hold their position and no sloughing occurs, we should here have a very good covering over the bladder. At a subsequent operation an attempt might be made to make a roof for the urethra from the newly-formed cover of the bladder and from the sides of the penis. If we get satisfactory union in this case, the child will be able to wear a soft rubber urinal and thus prevent the soiling of the clothing. These operations for exstrophy of the bladder are attended with a certain amount of mortality, due to shock and exhaustion following profuse suppuration ; but this may be materially diminished, if care is taken to keep the patient well wrapped up during the operation, to lose as little blood as possible during its performance, and to have him well surrounded by hot-water cans after the operation until reaction is well established. If the wound can be kept aseptic, repair is rapid and the risks of profuse suppuration are also avoided.

NECROSIS OF THE TIBIA FOLLOWING EPIPHYSITIS.

This little girl, six years of age, I bring before you to show an interesting inflammatory condition of the tibia. The history of the case is as follows. Several months ago she received a blow on the upper portion of the right tibia, which was followed by pain and swelling. This became so marked that she was confined to her bed and suffered from considerable fever, and in a short time developed several abscesses which opened over the anterior surface of the tibia.

When I examined the case, upon her admission to the hospital, I noticed that there was a marked deformity of the right leg, due to its being bowed outward ; the head of the fibula was very prominent, and there were also several sinuses over the tibia which were discharging a little pus. An examination of these sinuses with a probe proved that they led down to necrosed bone, and upon grasping the tibia I found that there was very distinct motion at the junction of the diaphysis with the upper epiphysis. The necrosis here has probably originated from an acute epiphysitis following the injury, and there is probably quite a large sequestrum present at this time. Epiphysitis, I am confident, plays a very important part in the production of caries and necrosis in the long bones. I propose to-day to expose the dead bone and remove it. The patient being etherized, and an Esmarch bandage being applied

to render the limb bloodless, I cut down upon the tibia from a point a little above the upper epiphysis, making an incision about eight inches in length, and expose the bone freely. Upon separating the periosteum, which is very loosely attached except at the inner portion, I find that the great bulk of the shaft of the tibia, for at least six or seven inches, is absolutely dead; a small strip of involucrum, or new bone, is attached to the periosteum upon the outer aspect. With an elevator and forceps I am able to separate this sequestrum and remove it, and find that it is perfectly detached at its junction with the upper epiphysis of the tibia. The cavity is then thoroughly curetted and washed out, and I find that there remains only a narrow strip of bone, mentioned before, upon its outer surface. I have no doubt that if this cavity is packed and allowed to heal by granulation, satisfactory recovery will take place; but I propose to-day, with a view of hastening the healing, and also lessening the deformity which results from these large cavities in the healing of bone, to introduce some bone-grafts according to the method of Senn,—that is, using the decalcified bone chips made from the fresh bone of the ox, packing the cavity with these, and then bringing the periosteum and skin together with sutures over this packing. You notice that I fill up this cavity very thoroughly with bone chips and then introduce sutures, and apply over the line of incision a large pad of iodoform gauze, applying outside of this a layer of bichloride gauze and several layers of bichloride cotton, and these dressings are held in place by a firmly applied bandage. The Esmarch strap is next removed, and after waiting for a few moments to see that there is no hemorrhage, I place the limb in a well-padded pasteboard gutter, to give fixation to the parts, and the child is removed to her bed.

In using bone-grafts, it is essential that the wound or the bone-cavity be rendered perfectly aseptic. If this condition is obtained the blood from the bone fills up the cavity and the interstices between the bone chips, and vitalization may take place very promptly. If such a result is obtained, we save much time in the healing and also at the same time lessen very much the deformity resulting from these operations for the removal of caries or necrosed bone. It has been my experience in some cases to have failure as regards vitalization of the chips, and in such cases they simply act as foreign bodies and they can be removed at a subsequent dressing; and after the cavity has been thoroughly irrigated, it should be packed with iodoform gauze and allowed to heal by granulation. I hope in this case that vitalization of the bone chips will occur, and that I shall be able to show the patient at a subsequent clinic as a good result from the use of bone-grafts.

FEMORAL HERNIA; TRAUMATIC FISTULA OF THE URETHRA; TUBERCULOSIS OF THE HIP-JOINT; TEMPORARY RESECTION OF THE HIP-JOINT.

CLINICAL LECTURE DELIVERED AT THE RUSH MEDICAL COLLEGE.

BY JOHN B. HAMILTON, LL.D., M.D.,

Professor of the Principles of Surgery and Clinical Surgery in Rush Medical College,
Chicago.

GENTLEMEN,—This patient, a cook by occupation, comes to us for relief from a tumor which projects below Poupart's ligament, and which has existed for several years. Sometimes she is herself enabled to press it back into the abdomen. There is a distinct impulse given to the tumor on coughing. It is ordinarily painless, but sometimes when tense becomes painful. She has been told that it is a hernia, and an examination proves that this statement is correct. It is a femoral hernia passing out of the crural arch of the abdomen and projecting into the saphenous opening. It is sometimes easily reduced, and there must therefore be a large ring. It reduces itself when she lies on her back. Occasionally there is a great deal of pain connected with it, and she is obliged to seek medical relief. She desires to have an operation performed for the radical cure, and that operation consists in the same general methods of treatment which are practised in the operation for the radical cure of inguinal hernia.

I desire to say a few words on the diagnosis of femoral hernia. Generally, the impulse given to the tumor on coughing is taken as a crucial test, that impulse being held to mean that the tumor is connected with the contents of the abdomen. There are other conditions beside hernia which give rise to that impulse,—for example, the accumulation of an enormous amount of fluid in the abdomen, of any kind, tubercular, for example, passing out through the saphenous opening, may give rise to precisely the same feeling or impulse that exists in the case of hernia. But we can reasonably exclude it in the diagnosis in this case. Having made that exclusion, we are satisfied that we have to deal with a femoral hernia which, if it be reducible and not strangu-

lated, the radical treatment is comparatively simple. If the tumor projects much, it is easier to find the sac than where the hernia is reducible and passes into the abdomen. Let us remember the relation of femoral hernia. A femoral hernia passes out of the crural arch to the inner side of the blood-vessel next to the femoral vein, passes along underneath the fascia lata until the opening which exists near the saphenous vein is reached; then, considering the patient as standing upright, the gut passes downward through the crural arch and forward through the saphenous opening, where it makes its external subcutaneous appearance.

For the reduction of these tumors, with our modern views as to taxis, we no longer believe it necessary to make long-continued efforts to reduce a strangulated hernia, or to reduce a femoral or inguinal hernia, believing that this manipulation sometimes produces extravasation. I have in one instance seen half a pint of blood effused on the outside of the hernial sac, because long-continued and vigorous efforts at taxis had been made before an operation was performed. Furthermore, taxis should be made gently; if the tumor cannot be reduced, operative means should be employed.

The operation for femoral hernia consists in making a straight incision over the anterior portion of the sac, continuing the incision directly down to the sac. Having reached the sac, we raise it with tissue-forceps and incise it; if there is no strangulation the gut is simply reduced by the fingers. If there is strangulation, we feel for the constricted portion with the index finger, while the other finger and the hernial knife divide it, the knife being usually directed upward towards the abdomen. That is done to avoid wounding the principal artery or femoral vein which lies to the outer side of the tumor. When the gut has been returned in this operation, we may then seize the sac itself and ligate it. We may fold or twist the sac on itself and suture it, or we may cut off the sac and suture it at the neck. The saphenous opening may be sutured or not, at the pleasure of the operator, but the walls of the opening should be sutured as near the femoral vessel as possible, and that may well be left to circumstances and the conditions as they exist. I would not lay down a hard-and-fast rule, whether or not to suture the saphenous opening; I would be guided by the conditions as found during the operation. Ordinarily I would sew up all openings which are not necessary for the traversing of nerves or blood-vessels.

An operation for the radical cure of hernia would be futile unless we removed the sac. You will observe that we have now reached the

sac, and it is, in fact, being forced through the saphenous opening. It is very thin; I incise it; a little fluid escapes. If I should find omentum, it might be proper to excise it along with the sac, especially if there are inflammatory changes. I will now introduce my finger into the sac, carry it directly backward until I reach the saphenous opening, then push the finger upward and feel the ring. I have the finger in the ring, and find that none of the contents of the intestine have protruded with the sac. The little omentum that was present was pushed back, so it will not be necessary to remove it. I will separate the sac, as much as may be, from the tissues, and take with it the attached fat, if necessary. I have now separated the sac as it projected through the saphenous opening, and, in order to make sure of it, just before I ligate I will put my finger inside again. I desire to secure the sac as close to the crural opening as possible. Here, you see, is the sac laid open, showing that its smooth lining is continuous with the peritoneum. The upper portion is thin. You see how difficult it is, where a hernia does not project, to determine the neck of the sac. I have now dropped the stump of the sac through the saphenous opening, and we shall close the femoral canal with curved needle and catgut. I shall use Wood's needle for this purpose, using my index finger as a guide for the operation. Between my finger and the needle I have the femoral vein, and by pressing towards the crest of the ilium I can feel the pulsation of the artery, and the vein lies just on the inside. I push my needle towards the pubes, and, for convenience, I shall bring the point out through the skin. I thread it and withdraw it; the needle is then passed through the other side. I now tie and tighten these ends. I have closed the ring so that the finger cannot enter it; it now remains to close the external wound. I might in this case insert a small drainage-tube, but the hernia is so small and the ring is of such small proportions that it is hardly necessary. There has been no hemorrhage or oozing, so a drainage-tube will not be required. These projections which you see are simply lobules of fat which project at the saphenous opening, and may be excised or not. They are not particularly in the way, hence there is no good reason for removing a part that is not concerned either in the disease or the operation.

TRAUMATIC FISTULA OF THE URETHRA.

The next case I show you is one of considerable interest; it is a case of traumatic fistula of the urethra. It has been my lot to encounter these cases very frequently among seafaring men, mostly as a result of falls from aloft, where the perineum received the force of

the fall in such a way as to bruise the tissues, which subsequently becoming infected, a fistula resulted. On one occasion I remember seeing a traumatic fistula of the urethra which was produced in this way: a farm-hand, sliding off a hay-stack, alighted on the end of a pitchfork sticking in the ground, and became empaled on the fork-handle, which had previously been broken obliquely. The resulting wound healed partially, but a fistula was left. I have seen a case of similar character in the person of a sailor in the Marine Hospital. These cases are very troublesome. Sometimes ischio-rectal abscesses are followed by urinary fistula. I have seen one such case at the Marine Hospital, where the urine dribbled into a large abscess-cavity; it was originally an ischio-rectal abscess, but was neglected, finally opening into the urethra.

In the case before us, the child was injured by the kick of a horse. There was some inflammation, an abscess, and probably rupture of the urethra; but whatever condition was present there was left a urinary fistula. On the day I first examined the patient (yesterday), I was unable to establish any connection between the penile portion of the urethra and the portion behind the scrotum through which the urine flowed,—that is to say, the anterior portion is practically cut off by the cicatricial tissue resulting from the wound. These cases are very troublesome, not only to the patient, but to the surgeon, inasmuch as they are difficult to cure by ordinary operative procedures. We would suppose that in such a fistula, with edges free like this, and after close apposition, we could unite the wound, leaving a catheter in the urethra and restore it, but practically we find that the bacteria in the urine reinfect the wound, and that it reopens sooner or later. In some aggravated cases which have existed for a number of years, I find that they can only be cured by performing a suprapubic cystotomy, so that the bladder is drained elsewhere, then operate on the fistula, and union takes place. I find very few instances where a simple union of the fistulous opening in the urethra is followed by success. However, other surgeons may have been more successful in performing the operation differently, but in some fifteen cases that have come under my observation the result has invariably been that the wounds did not heal by primarily operating on the fistula. It is a serious thing to perform either suprapubic cystotomy or perineal cystotomy in order to make a channel for drainage, if we can avoid it by resorting to other means.

I propose in this case to open the urethra fully, to overdistend it, to make it so that the urine will pass the obstruction for a few days by

the insertion of a catheter, and then try to unite the fistula over the catheter, which is to be retained in the urethra. I must say, however, that my previous experience does not warrant the hope of very great success; but, as we have a child to deal with, I will endeavor to make the operation in that way, because it is a less serious operative procedure than opening the bladder would be, and perhaps we may succeed; if successful, it will obviate the necessity of suprapubic cystotomy.

[The child was here etherized.]

In order to facilitate the introduction of the instrument I shall fill the urethra with oil. You will notice here an opening that exists in the urethra just below a scab in the median line. I can feel a hard infiltration about that region. I pass one of these whalebone guides as far as it will go, on account of this change in the direction of the urethra, with the hope of passing the guide on into the bladder. You will see that, instead of going into the bladder, it passes out through the lower opening of the canal. We will reverse the point of the instrument and see if I can turn it; or, if I can pass another into the bladder, I shall be satisfied. I use oil in preference to vaseline because it is a much better lubricant for the urethra and less irritating. I shall pass the instrument down until I reach this junction. I have not succeeded in what I wished to do, and I shall therefore use another kind of instrument, a steel bougie. If I fail to introduce this into the bladder I shall make an opening in the perineum and open the bladder through it, enlarge the original wound, at the same time keeping the canal in the anterior portion of the urethra open. I shall incise the old wound, keeping on the "gripe." I must avoid the bulb, which is very near the upper end of this incision, keeping the knife directly in the median line. The cicatrix here is in the line of the incision. It is astonishing sometimes to see to what extent cicatricial tissue will form about the urethra. I have now reached the end of this instrument, and shall endeavor to establish communication between the anterior portion of the urethra and this opening. It is apparent that it must be preliminary to effecting any cure. I have only established a communication between the anterior portion of the urethra and this wound. If I cut through the wound and make an entrance into the bladder, it will have to be done now. By putting the child in the lithotomy position we will then have as favorable a condition as for an ordinary lithotomy. I now put my finger within the anus in order that I may have a guide to the direction of the bladder and the pubes. Sometimes the urethra lies close under the pubes, and it is not always

easy to reach it. I shall also observe from what direction the urine comes when I make pressure on the bladder, so that we may cut out this fistulous tract if it exists outside of the urethra. I now have the instrument in the bladder, because the beak turns freely in all directions. I now dilate the urethra as fully as possible in a child of this size in order that I may pass a soft catheter. I find I shall have to enlarge the opening. When a larger dilating instrument is passed there is obstruction, which of course marks the site of the original cicatrix. It is very dense, firm, and it is almost impossible to force an instrument through, so I am obliged to make a much more extensive incision than I now have. I shall expand it so that I shall have no difficulty in putting a catheter through it. I have expanded it considerably, and I shall endeavor to keep the channel I have made open. The catheter is now in the bladder. I believe with the opening I have made into the bladder I can introduce a larger catheter, but it will be hardly worth while. For a few days most of the urine will pass out through this wound alongside of the catheter. The case is in the condition now of one of perineal section or lithotomy. I will close up the lower angle of the wound with a couple of sutures. One can scarcely imagine a more difficult case of urethral surgery than this, and any one of experience will understand what those difficulties are. A good many times I have passed an instrument into the bladder, made a pretty large opening, but found afterwards that the introduction of a drainage-tube was attended with a great deal of difficulty. Operating in the median line, the perineal muscles act as sphincters to this wound and partially close it; it is for that reason that the instrument in attempting to enter through a recent incision sometimes passes off in other directions. I shall leave here secondary sutures in order that the wound may be closed after the withdrawal of the catheter, if it is deemed expedient. The wound will now be dressed. I expect the wound will close as an ordinary lithotomy incision would do, and still retain this channel through the urethra. If we can maintain the calibre of the anterior portion of the urethra, we may expect a cure; but, remembering the statistics of this operation, I have little promise to hold out.

[NOTE.—The contraction returned in about four weeks, and suprapubic cystotomy was resorted to, and the urethral fistula was closed by sutures over a soft bougie.]

TUBERCULOSIS OF THE HIP-JOINT; TEMPORARY RESECTION OF THE TROCHANTER.

I have not recently examined this child, and I do not know, therefore, what operation will be performed, but we will find out as soon as the examination is completed.

She has been in the hospital several times; sometimes having extension apparatus applied, at other times remaining in bed with weight and pulley. But all of that kind of treatment, which is simply palliative and intended to hold the joint surfaces apart while the child is under tonics and other means of treatment, has been without avail. She now comes back to the hospital unable to walk, unable to move the hip without severe pain,—so painful has the joint become, in fact, that I propose to conduct the examination under anæsthesia, and then proceed to perform whatever operation is required and can be done at this time.

On turning the child on the side, you will notice a fistula posterior to and a little below the trochanter. We will take the probe and ascertain the direction of the fistula. I find that the probe passes directly into the joint. There is, therefore, only one way to treat this joint successfully at this stage,—that is, to make an opening into it, remove either the soft parts by arthrectomy, or by resection of the bone, or both, according to the extent of the disease. If this girl were an adult instead of a child, there would be no objection to performing a typical resection, but in children an atypical one should be the rule.

I will content myself by cutting down upon and performing a temporary resection of the trochanter. We simply cut down upon it by a linear incision, divide it with the chisel, lift it up with its muscular attachments until the neck of the femur is fully exposed. We are then able to reach the joint. I make my incision directly over the trochanter, slightly curved, but passing deeply down to the bone. I have separated the soft parts from the bone as much as is required, in order to make a linear incision to the trochanter. I have separated the trochanter from the femur with the chisel, and the incision is directly on a level with the upper surface of the neck of the femur. I shall turn the trochanter directly upward, which gives complete access to the capsule of the joint. I now separate the neck of the femur from its attachments, open the capsule, and expose the joint. Now, by rotating a little I can put my finger directly into the opening and feel the amount of erosion that exists, with the head of the bone still within

its socket. If it be not too great, I prefer to do but little else than to make an opening into the joint and provide for the injection of iodoform emulsion.

Remember what I have previously said about resections in children. We avoid them whenever possible, as we stop the growth of bone; whereas, if we can perform an arthrectomy, or, by gouging out the carious portions of the bone, preserve the joint, we will have accomplished very much more for the child.

I find a sinus with a roughened surface on the face of the bone, and I shall gouge out this opening first, as the bone is quite soft. I shall be able to do it without using the knife. This is being done subperiosteally. I am passing a gouge longitudinally along the neck of the femur to the head of the bone, and removing the carious surface, which I will soon have complete access to, piece by piece. The pus, which is very plentiful here, exudes from the bone in several places, and we have evidence of its commencement in the medulla of the bone and its cancellous structure. I find that the acetabulum itself is involved, and a rim of the acetabulum will have to be scraped out. This I am doing. The process is tedious. In the removal of these fragments of bone you will find it expedient to use the finger as a guide, keeping it on the fragment while the instrument (the sharp spoon) is being fixed. In this way you avoid unnecessary damage to the soft parts, and it enables you to have control of the operation at nearly all of its stages. The specimen which I show you, as now removed, consists of half of the side of the neck of the femur and half of the head. You will see how the disease has progressed around the head of the bone. The white, glistening portion is the articular surface of the head of the femur; the eroded portion of the other side is where the tubercular erosion has extended into the head of the bone. We now irrigate the joint, as there is a great deal of eroded surface; the wound is dusted with iodoform powder, and, as before, iodoform gauze is inserted for drainage. There is a sinus underneath the bone in the soft parts, extending into the cavity of the acetabulum, which I thoroughly scrape with the sharp spoon. I now bring the trochanter back into its place, leaving a drainage of gauze from the most dependent portion of the wound which passes directly into the joint. I sew the trochanter into position with very heavy catgut. I pass a needle through the periosteum, the fascia, and sheath of the muscles attached to the trochanter, and through the same structures on the shaft of the femur, this bringing the parts into perfect apposition. The power of rotation of this child's thigh when the trochanter and bone unite, which will be

in about three weeks, will be as perfect as before the operation. Care should be taken that no tension is put upon the stitches that can be avoided. The limb should be fixed in a position which brings the bone together without tension. The packing which is being inserted is in the intermuscular septum, where the pus formed during the progress of the disease, and which has been thoroughly scraped and irrigated. You will notice there has been very little hemorrhage. Hemorrhage is much less when the trochanter is resected than in the other operation.

The dressing in this case will be precisely as usual in these cases. The parts about the wound are to be well cleansed, iodoform gauze and antiseptic absorbent cotton applied, and a plaster cast over all. The pelvis must be fixed to secure immobility at the hip-joint.

The history of these cases after resection of a joint, performed as this was, is generally one of uninterrupted convalescence. Occasionally we have a sinus formed directly in the tract of the wound, due, undoubtedly, to some pyogenic infection, some bacterial colonies which were left in some undisturbed pocket or side-track at the time of the operation. But in the majority of cases we do not expect any result other than a steady progress towards uninterrupted convalescence. It is a crippled limb, but it was this before the operation was performed. We have stopped the pus formation; more than that, it differs from an ordinary resection in this particular: after the old methods of resection, the limb hung like a flail; while it might be as powerful as before, yet the power of rotation was completely lost; and, in case of resection of the head of the humerus, in many instances the power of elevating the elbow to the level of the shoulder was lost. By temporary resection of the trochanter in the femur, and temporary resection of the tuberosity in the humerus, we preserve in these limbs respectively the full power of rotation as perfect as before the removal of the head of the bone.



FIG. 1.—Exophthalmic goitre. (Before operation.)



FIG. 2.—Exophthalmic goitre. (Three months after operation.)

THYROIDECTOMY FOR EXOPHTHALMIC GOITRE; ENUCLEATION OF AN ADENOMA OF THYROID GLAND.

CLINICAL LECTURE DELIVERED AT ST. LUKE'S HOSPITAL.

BY B. FARQUHAR CURTIS, M.D.,

Surgeon to St. Luke's Hospital and to the New York Cancer Hospital.

THYROIDECTOMY FOR EXOPHTHALMIC GOITRE.

GENTLEMEN,—Our patient is a young woman of twenty-four years, who enjoyed good health up to three years ago. She has menstruated regularly and normally since the age of fourteen. Three years ago she noticed that her neck was becoming prominent, and about the same time nervous symptoms of various kinds made their appearance; for instance, the heart began to beat very rapidly and the eyes became prominent. The goitre—for that is what was causing the prominence in her neck—grew slowly, but no treatment of importance was resorted to until about one year ago. At this time the neck measured thirteen inches. Since then, under a thorough course of treatment, extending over several months, there has been a reduction in the size of the tumor and an improvement in the other symptoms. During the summer her general health improved, and there is now none of the insomnia which was a marked symptom in the earlier part of the case. But the improvement has ceased, and marked nervousness, with a pulse of one hundred and forty per minute, persists and requires relief.

The treatment of exophthalmic goitre by medicine and electricity is effectual only in the comparatively mild cases. In a recent collection of twenty-nine cases in which surgical treatment (thyroidectomy) had been employed, twenty-three were reported cured, and three improved; one died, and the others were lost sight of. This report is exceedingly favorable, for if we can cure seventy-five per cent. of these cases in which medicine is of no avail, it is well worth while to give the surgical treatment a trial. The risk of the operation is no greater than in ordinary cases of goitre; there is no more hemorrhage

or shock, although there have been a few more cases of sepsis reported. This is probably only an accidental complication, and not inherent in this form of goitre.

The way in which a surgical operation benefits these cases is very obscure, for we do not know the exact nature of the disease. We know, however, that galvanism of the sympathetic will cure a certain number of cases, and we also know that the thyroid is intimately connected with the sympathetic, and that a wound made in removing it may divide some fibres or may affect the nerve more or less by the process of healing, even if the nerve itself is not attacked. It is rational, therefore, to suppose that by some nervous effect—by actually dividing the nerve or by some derivative action—the cure is obtained. There is one theory of the pathogenesis of exophthalmic goitre which supposes the disease to be due to unnatural activity of the gland and too great a quantity of its vital products entering the circulation. Should this prove to be well founded, it would at once explain how removal of the gland would cure the disease. The operation differs in no way from the ordinary operation of thyroidectomy.

Our patient has a well-marked tumor; the neck is very large, rather more so on one side than on the other, the right half of the gland having attained the size of a hen's egg or larger. I shall make a vertical incision from the sternum to the cricoid cartilage, and then upward obliquely, turning down a flap which will expose the right half of the thyroid. The superficial veins will require to be secured as we proceed; two or three of the larger ones can be seen before making the incision. It is necessary to make quite a free incision in these cases, because thorough exposure of the gland is most important to allow of careful dissection. The principal danger of the operation is from the hemorrhage from the numerous vessels and from many minute bleeding-points which are very difficult to secure. There is also some danger of injury to the recurrent laryngeal nerve. Every vessel should be secured between double ligatures, rather than by leaving clamps hanging to the vessels, owing to the great number of these instruments which would soon accumulate in the field of operation. We have now passed through the first layer of vessels, and the muscles of the neck—the sterno-hyoid and sterno-thyroid—are divided vertically in the line of the original incision. The tumor can now be seen protruding in the wound, and you notice the large thin-walled veins which course over its surface. The tumor is now drawn from the median line, and I gently tear my way through the fascia, keeping a sharp lookout for the vessels which should pass off from the superior horn.

It is not important to separate the veins from the arteries for ligation, for the veins usually have such thin walls that they can be safely secured in the ligature which confines the artery. There is usually an accessory vein besides the vein which directly accompanies the superior thyroid artery, and it is possible that the vessel which we see lying in the wound now, is this one. The veins are rather uncertain in their arrangement here, and they must be tied as they make their appearance. Having dissected well down on the posterior surface, we shall continue the dissection farther on the outer side, and then work more deeply. In passing ligatures it is wise to have some systematic method of holding the aneurism needle, so that the ends shall not become twisted. I usually hold the thread from the concave side of the needle in my hand, and picking up the loop on that side also when the needle has been passed under the vessel, the thread cannot be twisted, for the twist is removed by the needle straightening out the other end as it is withdrawn. You can see now the inferior thyroid vein—an important vessel—as it empties directly into the innominate, which is not very far off. At the lower and outer corner of the gland is a nodule which extends directly downward into the chest. We must try to get rid of this first in order that we may carry our dissection closer to the tumor, and so avoid injury to the nerve. I shall now dissect upward, keeping between the fibrous capsule and the gland. The treatment of the isthmus is upon the same principle as that of a pedicle on an ovarian or other tumor; it is transfixed and ligated in two parts. A double ligature is passed, crossed, and secured with a knot. The two ends are then ligated over one-half of the pedicle, and the other two ends tied over the other half of the pedicle. You see that the tissue is soft, and the ligature readily sinks into it. The tissue which you see has been left covering the trachea is the posterior part of the fibrous capsule of the gland. The cutting and tearing and the application of the ligature were on the outside of this tissue, and hence were done at a safe distance from the recurrent nerve. The wound appears to be absolutely dry, so that drainage does not seem necessary. The muscular planes will be held together by a few interrupted sutures of catgut, and the flap kept in place by a continuous catgut suture. I think it is an advantage to suture the divided muscles and the deeper fascia, because the natural layers of the neck are restored, and there is therefore less likelihood of a depression being left to mark the former site of the tumor. No drainage-tube was used.

A doubt has been raised recently as to the influence of the complete removal of the thyroid in producing cretinism. The result of experi-

mental work on this point is not quite uniform. The cases where complete removal has not been followed by cretinism are possibly explained by the presence of a small accessory thyroid, such as we found in this case, which has been left and been sufficient to prevent the nervous disturbances which are the rule after complete extirpation.

The dressing of these cases is very important. The region is difficult to cover in, and it can only be done by enclosing both the shoulders and the head in the dressing. I am putting on a pretty firm dressing, because if the parts are not well supported there is considerable risk that vomiting and coughing may displace the ligatures and give rise to secondary or, more strictly speaking, intermediate hemorrhage. A number of such cases have been reported.

The portion of gland removed, comprising the entire right lobe, shows on section a great distention of all the follicles, appearing not unlike a section of normal thyroid tissue under a low magnifying power. The pathologist reports "normal thyroid tissue."

ENUCLEATION OF AN ADENOMA OF THE THYROID.

I will now show you a second case of enlarged thyroid, but of a different kind, upon which I propose to operate to-day. This woman, H. F., is married, and is thirty-one years of age. Previous to the present trouble she enjoyed excellent health. Six years ago she first noticed a small hard lump in the front of the neck. This tumor has never been painful, nor has there been any trouble with her eyes or any cardiac palpitation or dyspnoea, but she has distinct "roaring" when she breathes quickly. The growth of the tumor was quite gradual up to six weeks ago, but since then it has been quite rapid, and its increased projection has been particularly noticeable.

The patient presents a perfectly globular mass the size of a large orange, semi-fluctuating like a tense cyst, attached to the thyroid, but lying directly in the median line of the neck; but it is impossible to say from which half it originates. It is tense, semi-fluctuating, but we hesitate to pronounce it actually a cyst.

The benign tumors of the thyroid gland may be roughly divided into the adenomatous, parenchymatous, and cystic. True adenoma is rare, if we understand by that term a tumor in which there is not only an increased number of follicles, but a proliferation of the epithelium within them. The parenchymatous tumors on section represent exactly the structure of the normal gland. The growth may seem to invade the entire thyroid, or may be distinctly encapsulated, probably it is always encapsulated, but has enlarged so evenly as to replace one-half



FIG. 3.—Enucleation of goitre. (Before operation.)



FIG. 4.—Enucleation of goitre. (After operation.)

of the entire gland, the substance of the latter being expanded into a very thin encapsulating layer, which can scarcely be recognized as glandular tissue. It is this encapsulation which makes feasible the methods of enucleation, one of which I propose to employ in this case. The cystic tumors are simply immensely-distended follicles filled with serum, or more frequently a colloid material. They are also encapsulated, and can be easily enucleated. Both parenchymatous and cystic tumors may be single or multiple.

The patient is now in position, with head and shoulders well elevated. An incision is made in the median line over the most prominent part of the tumor, from the sternal notch to the thyroid cartilage, and it is then ascertained that the tumor has grown in the left half of the gland, displacing the trachea in a wide curve to the right. The incision is then enlarged by another, extending from its upper end obliquely outward and upward beyond the limits of the tumor. Without dissecting back the flaps the incision is deepened to the true capsule of the tumor, and then it is partially shelled out of the loose connective tissue until the finger can be carried beyond the greatest diameter of the tumor.

I do this to secure a hold for the temporary rubber ligature which I propose to apply. This device, suggested some years ago by Rose, to prevent hemorrhage, has answered very well in my hands in the enucleation of such tumors, preventing all hemorrhage while the ligature is in place, and not visibly increasing the loss of blood during the latter part of the operation.

While my assistant lifts up the tumor somewhat from its bed I make two moderately-tight turns around its base (inside of the skin flaps) with a piece of elastic one-fourth inch rubber drainage tubing, and secure it with a knot. A careful incision is then made through the capsule, and when the surface of the tumor itself is reached the capsule is stripped back from it with the fingers. Care is necessary to actually reach the surface of the tumor before beginning the enucleation, as it will otherwise be impossible to accomplish it, and it is often very difficult to recognize the tumor tissue. As the tumor is shelled out, assistants must seize the edges of the capsule and hold them out on all sides in order to prevent the ligature from suddenly slipping off when the tumor is removed.

The patient suddenly breathes very badly, becomes very cyanotic, and it is evident that there is some obstruction to the passage of air. This is not relieved by drawing the tumor forward, but when I finish the enucleation rapidly, pack the cavity with sponges, and remove the

rubber ligature, she recovers herself at once. The trachea is not softened, and it seems scarcely possible that the ligature could have caused pressure enough to close it, but perhaps as the tumor was enucleated the soft parts below were drawn into the grasp of the ligature, and there was some pressure upon the recurrent laryngeal nerve.

[The sponges were now removed from the cavity and all bleeding-points secured by fine catgut ligatures. There was such profuse general oozing, however, from the inner surface of the capsule that it was considered necessary to pack the cavity with iodoform gauze. The edges of the wound were drawn together over the gauze and secured by a few temporary silkworm-gut sutures, and the usual dressing was applied.]

A section through the mass removed shows that it has the usual structure of the thyroid gland, and the microscopic examination confirms the diagnosis of parenchymatous goitre.

[NOTE.—Both cases ran a good course, the first leaving the hospital with absolute primary union in about ten days, and with great improvement of all her symptoms, even in that short time, and the accompanying picture, taken three months after, shows how great this improvement is. The nervousness has disappeared and the pulse-rate averages about one hundred per minute, without medication, rising only to one hundred and twenty on exertion. The patient sleeps well and has gained in weight. Nine days after the operation the packing was removed from the wound in the second case; the edges were freshened and closed by silk sutures. Primary union was then obtained. The picture shown was taken six days after the insertion of the sutures.]

Genito-Urinary and Venereal Diseases.

THE DIFFICULTIES OF DIAGNOSIS OF SYPHILIS IN WOMAN.

A CLINICAL LECTURE.

BY ALEX. RENAUL, M.D.,

Physician to Broca Hospital, Paris, France.

GENTLEMEN,—At first thought one would say, Why is there any difficulty in such cases? Are not the manifestations of syphilis the same in man and woman? or is the evolution of the malady different in woman? Surely the symptoms have been often described, and any physician should have no difficulty in making a diagnosis of syphilis in woman.

In principle this is all true, but in point of fact there are a number of difficulties that should be mentioned. It will be noticed that syphilis is generally found in man, while it seems to be relatively rare in woman, at least in the first stages of the disease, so that if one is called upon to see several such cases difficulties will arise that will show that the diagnosis is not at all the easy one that it is in males.

Three causes obscure the diagnosis of syphilis in woman,—first, the conformation of the organs in the female sex; second, the character of the symptoms; third, the insufficient corroborative facts. I do not need to describe the external organs of generation in woman, but allow me to say that the vulva is composed of three planes,—a superficial one, in front of the mons veneris and behind the labia majora; a middle plane, represented by the labia minora and the clitoris; a deep plane, the vestibule, meatus urethræ, and the vaginal orifice. These all cover one another so closely, particularly in women who have had no children, that they are almost one body, and that fact is a source of great difficulty in the diagnosis of the first sign of the disease, just when it is most important to find it. The chancre may be found on the labia majora, and there the difficulty is slight, but this is not at all its most common seat; the specific ulcer is frequently hidden in the fold that lies between the two vaginal lips, under the hood of the clito-

ris, at the entrance of the meatus urinarius, or, again, behind the labia minora, especially when these are large and pout out like what is called the Hottentot woman's apron. Again, it may be behind the fourchette or in a crease by the carunculæ myrtiformes; finally, the chancre in woman may be very small or even have a linear form, which increases the difficulty of diagnosis.

From these facts to the precept, that one must pay the greatest attention in making such an examination, is only a step: every fold of the vaginal mucous membrane must be examined with care, the meatus urinarius must be opened, the fourchette pulled down, and the caruncles looked to; finally, a speculum must be introduced, for the chancre may be found on the os uteri; this I saw several times last year. The lesion may also be discovered in the vaginal canal itself, although this is rare.

I have said that the second cause of difficulty in diagnosis is the character of the symptoms (these are called "*fruste*" by the French, meaning obscure or worn out). The initial accident, the chancre, which is so characteristic in man, is not so in woman, in whom it is not hard nor painful, and may, indeed, not be found at all. I have constantly looked for this characteristic and have rarely found it hard in woman. Once or twice I saw it so, close to the clitoris and the meatus urinarius, but as a rule chancre in woman is simply a parchment-like appearance of the mucous membrane, which will escape a superficial examination.

Professor Fournier has given a method to find it. "Seize," he says, "with two fingers the extremities of the largest diameter of the ulceration and raise it up as though you would tear it off, and you will get the parchment-like sensation these chancres give." This is the best method to find the lesion in woman.

Why is it that the induration, that is almost pathognomonic in man, cannot be detected easily in woman? It is because this anatomical state does not depend upon the nature of the ulceration itself, but upon the structure of the parts beneath. It is important to state this fact: that any inflammatory neoplasm, specific or not, will be all the harder the more difficult it is for the inflammation to propagate itself. These conditions are found in regions where the skin is directly adherent to the adjacent parts without any cellular tissue to speak of underneath which will allow of its diffusion. The hard chancre is most frequently found in man just in the groove we call the "balano-preputial," because here it lies on the elastic fibres that constitute the larger part of the gland. In woman, on the other hand, the genital chancre is found

mostly on the labia majora or the labia minora, or in the fourchette, just in such places that induration could not take place.

So how can one distinguish a chancre from herpes of the lips, a simple vulvitis, or even a slight scalding? There are, fortunately, some points to go by; the first and most important is that a syphilitic chancre is generally *single*. It is indeed very rare to find more than one erosion in syphilis. The next point is œdema of the labia majora. When you examine a suspected woman and find an œdema of the labia majora, you may be almost certain that there is a chancre there, or, at least, a cicatrix from it. The next valuable point is adenitis. This follows a chancre, as Ricord said, "as the trunk follows the root of a tree." This is a unilateral hardening and swelling of the ganglions that is characteristic. It is hard and indolent, thus differing from the adenitis of chancroid, which is painful. In ulcerated herpes there is no adenitis, and in vulvitis, when it exists, it is bilateral, appearing on both sides.

When we come to secondary symptoms of syphilis in woman it is not so difficult to make a diagnosis, as a rule, but many are astonished to find that it is not so easy as in man, under certain circumstances. Let us first run over the usual signs that we find in such cases. On the skin there should be roseola, or a series of red or rose-colored marks; then the hair is thinned out and some crusts are found on the head; looking at the pharynx and the tonsils we may find some grayish plaques which are accompanied with some dysphagia, while around the vulva and anus some mucons plaques will be met, and, finally, the inguinal, cervical, and epitrochlear ganglions show by their hypertrophy that the whole economy is invaded. Now let us return to these symptoms: the roseola may be so light that you cannot see it, even by turning them to the light. The late Dr. Quinquaud gave us a good method to use in such cases,—that is, to make the patient take a hot bath, when the rose-colored marks will appear. Again, a certain quality of skin in many women will fool you; these are of the numerous scrofulo-tubercular race; they have a marbled skin about the abdomen and limbs that looks very like roseola; but close attention will prevent mistakes; for instance, these rose-colored marbled skin marks are not isolated but continue in long bands, and are on the lower abdomen, thighs, and legs, while real roseolæ are under the breasts and on the flanks. Then, in case of syphilis roseola is not the only symptom you will find: in exploring the ano-vulvar region and the bucco-pharynx and the head, you will be sure to get some other sign; notice particularly the back of the neck just at the junction with the hair,

where you will often find some small copper-colored plaques that will lead to the proper diagnosis.

I repeat that the scrofulo-tubercular women that may have the rose-marbled skin should not give you trouble. Women of this type are easy to distinguish, as a rule; they look strong and have large hands, with a wine-colored face that seems like health, but it is far from it. If the roseola is scanty, only a few marks, that you find on the chest, examine the hands, in the palms, and on the soles of the feet, where some copper-colored marks will help you out.

At a more advanced period of syphilis you will find a special sign in women that appears rarely in men,—that is, the so-called “pigmentation syphilide”; it is really only an exaggeration of the natural pigmentation of the skin, and is found about the neck. This abnormal pigmentation sometimes takes a lace-like form, showing little islands of pigmentation and clear spaces between. This is almost pathognomonic, as it is not seen in any other malady around the neck.

As to the hair, the alopecia in women is sometimes very slight and you will not find anything, for many of them will have had seborrheic eczema and be losing a little hair, so if you stick to this one symptom the problem will not be solved; however, the little crusts (papules) on the surface of the skin of the head will help a great deal if found, as alopecia and these together hardly exist without syphilis, and the two have a considerable clinical value. Professor Fournier says that if this is complicated by a slight alopecia of the external portion of the eyebrows, it is a certain sign; this can be seen sometimes at a distance. The examination of the mucous membranes in women may not give you any sign; this is rare, though, for their mucous system is extensive and generally shows some signs of syphilis when it is present.

It is not so easy to judge of the color of the fauces, the pharynx, and the tongue. In the normal state the tint varies much: in some women the throat is always reddish and in others grayish, so that you must not be sure that the descriptions given in the classical books on the subject will be absolutely correct, and that you can go by them implicitly. They will tell you that the throat is gray in color in the secondary stage of syphilis; but a simple gray color will by no means indicate syphilis; it must be gray patches, which differ from the coloration of the rest of the mucous membrane of the part. As to pain, they do not by any means all have it: some women do not know that there is anything the matter with their throats, so that this symptom is not at all a sure one; indeed, it is often best not to pay any attention to the throat, but seek elsewhere for signs to guide you. The examina-

tion of the tongue helps sometimes when you find the border folds more creased than usual, but this is very indefinite: of course, when you find papules or plaques on the back of the tongue there is no longer any doubt, but if you are not accustomed to examining the tongue do not take the very large calyciform papillæ at the base of the tongue for syphilitic papules. It has been done, for they are extremely large in some patients.

There are some little white islands of sclerosis that are found on the borders of the tongue, looking like tumors of the organ, that are important in this diagnosis. The white plaques seen in smokers, as a rule, are found at the commissures of the lips or on the front, side, or tip of the tongue; they are triangular in form, while the syphilitic plaques are rounded and farther back; however, by the abuse of tobacco sometimes the sides and even the cheeks get these smokers' plaques, so this habit must be first inquired into. As to swollen glands and ganglia in women, it must be remembered that the sex are naturally lymphatic, and you will have the greatest difficulty in deciding if the adenitis you find is only scrofulo-tubercular or syphilitic. The ganglionic system in women is always much more developed than in man, and these signs have by no means the same value in them. The epitrochlear ganglion is an exception, and if you find it swollen the sign is of value, but it is rarely found in man even.

As to tertiary syphilis, the signs and symptoms are much the same in both sexes, and I will not dwell upon them except to say that in women the copper color of syphilides is important, and that in visceral syphilis they have much pain at night. You should also remember the importance of the aggravation of the symptoms when mercury is given by mistake in cancerous cases, which are or may have been taken for syphilitic ones. This therapeutic proof is worth trying in certain cases.

Finally, I must say something upon the third cause of difficulty, which I said was found in making the diagnosis of syphilis in woman,—that is, the insufficiency of the corroborative facts. It is extremely rare that a woman can give you any facts in regard to the incubation, the commencement, or the evolution of the malady. If they are town women they do not have the slightest idea of how it came about or when, and married women are almost as bad, as they cannot give you the slightest data. Therefore, do not count upon getting any information as to the *début* or evolution of this disease in women, as you can often in men. The women will say that they do not remember anything more than a slight form of inflammation, and perhaps a pim-

ple, but, as this may have come from some cause unknown to them, they usually think menstruation, they cannot tell you much, if anything, about it. The majority of them will deceive you purposely; the town women fear that if they tell it will get them into trouble or depreciate them in some way, and the good women are so ashamed of the thing having come from their husbands that they will not tell you anything; so, as I said before, you will get little or no information by questioning women about this disease.

When you cannot get any definite sign and suspect syphilis, you must, under some pretext or other, proceed to examine them all over; see the entire skin surface, examine the lymphatic system, the mucous membranes, and every portion of the woman. If, notwithstanding all, you cannot discover anything, try by adroit questioning to see if you can find out anything; remember that Beaumarchais correctly described a woman in saying "her natural instinct is to deceive," and unless you get her to describe her symptoms, without allowing her to suspect what you are trying to find out, you will fail. Failing to get a diagnosis give some simple remedy and make the patient come from time to time, and you will often be surprised, at last, by getting the signs you were looking for. It is important, however, to do this as soon as possible, as the specific treatment ought to be commenced early to succeed well.

DYSURIA.

CLINICAL LECTURE DELIVERED AT THE LONDON HOSPITAL.

BY CHARLES H. RALFE, M.A., M.D. (CANTAB.), F.R.C.P.,

Physician to the London Hospital, etc.

GENTLEMEN,—The subject which I propose for our consideration this afternoon is one which generally claims the attention of the surgeon rather than the physician. Nevertheless, when we eliminate the conditions which may cause a difficulty in passing water, such as a stricture of the urethra, an enlargement of the prostate, the formation of calculi or tumors of the bladder, etc., there still remain a number of morbid conditions, either accidental, local, or symptomatic, which call for the attention of the physician. If any of you are curious in such matters, on referring to the literature of thirty or forty years ago you will find frequent reference, in essays, lectures, and even monographs, to a condition known as *irritable bladder*, under which convenient and comprehensive term all causes which were not strictly surgical, and due to palpable lesions of the genito-urinary tract, were grouped, as explaining the less palpable conditions that induced dysuria. Although our advance in pathological and clinical medicine, especially in the chemistry of normal and diseased urine, as applied at the bedside of the patient has elucidated many of the obscure problems which were formerly so conveniently disposed of under a single term, still the study of bladder troubles from a physician's stand-point has not received that attention which such a varied and important subject deserves. This neglect is probably due to the brilliant success which has attended the labors of our surgical colleagues during the last decade in this department of surgical science. In spite, however, of this apparent want of attention, I shall endeavor to prove to you that a very considerable advance has been made during the last ten or fifteen years in this branch of pathology, and that we can now assign definite reasons for most of the conditions which produce an *irritable bladder*. In the majority of cases, also, we may entertain the hope of removing the disturbing cause.

What are we to understand by the term *dysuria*? The Greek prefix *δύς*, as applied to the terms dyspepsia, dyspnoea, dysentery, or dysuria, does not imply pain, but rather trouble or difficulty, a sense of hardness or the like. So that the older physicians who were very careful in selecting their phraseology, spoke of *dysuria* as only a simple difficulty of making water; *strangury* as an extreme difficulty, and *ischuria* as an absolute inability to pass any water at all. All three conditions expressing only so many degrees of a variety of morbid states, the prominent feature in each is a difficult or obstructed discharge of the urine. Without venturing on so minute a classification, I shall include the three degrees under the same head simply,—*dysuria*. An obstruction of the flow of urine either with or without pain. Indeed, we can hardly say any difficulty in making water is entirely without some sense of pain; either from a sense of resistance barely amounting to more than discomfort, to intense intolerance with frequent and scalding micturition.

Among conditions other than those arising from surgical lesions, or diseases of the viscera affecting the bladder, such as uterine tumors, pelvic disorders, etc., which are likely to cause this symptom, may be mentioned: I. Alterations in the quality of the urine. II. Morbid changes in the genito-urinary tract (not purely surgical). III. Disturbances of innervation.

I. CHANGES IN THE COMPOSITION OF THE URINE.

Of all the conditions leading to the establishment of dysuria, *alteration in the quality of the urine* is the most obvious and the most frequent. It may cause it by changes in its reaction, its specific gravity, and also by the presence of abnormal products, thus:

(a) *Highly Acid Urine*.—Simple excess of acidity of urine will cause urgency and frequent micturition. Some of you will remember a case in bed No. 8 (Charlotte ward) of a woman aged thirty-nine who was admitted under the supposition that she was suffering from stone. Examination proved that there was no morbid condition present in the genito-urinary passages, but that the urine was scanty, frequent and irritating, and had a high degree of acidity. Ordered to take three ounces of distilled water every three hours during the day, and a mixture containing twenty grains of citrate of potash three times in the twenty-four hours, the dysuria shortly disappeared and no symptom of urinary trouble remained when she was discharged. There are other instances of dysuria arising from a highly acid condition of the urine which are often more difficult to deal with, associated as they are

with intense acid dyspepsia, which Chomel¹ has described as "*la dyspepsie acide grave*," and to which Dr. Wilson Fox² also alluded, in which the whole body seems to turn acid. In cases of this kind the urine becomes extremely scanty, almost of a cherry color, rarely depositing uric acid or urates, free from albumen, but highly acid. The micturition is frequent and scalding and it is extremely difficult to give relief.

(b) *Alkaline urine*, whether due to a fixed or a volatile alkali, does not in itself seem to cause dysuria; when present, it seems to be caused either by the irritation of the deposited calcium phosphate in the one case, or triple phosphate in the other, both of which we will consider under the head of phosphaturia when we come to discuss the abnormal products in the urine.

(c) *Specific Gravity*.—Undue increase of the specific gravity of the urine certainly causes increased frequency and urgency in passing water. In cases under observation with a twenty-four hours' excretion of only thirty ounces of a urine ranging from 1025 to 1035 specific gravity, with no abnormal excess except of urea, patients have voided urine eight or ten times a day in quantities not exceeding two or three ounces at a time and always with a sense of urgency. In cases of low specific gravity increased frequency and urgency have been noticed, but in these cases it is difficult to say whether this may not be caused by an increased flow, or to reflex irritation from the kidney with which this condition is often associated.

(d) *Abnormal Products in the Urine*.—With highly acid urines, both *uric acid* and *urates* are often precipitated, though not in excess. Frequently they are the cause of the acidity from being eliminated too abundantly. The form of dysuria they give rise to is generally characteristic. There is great urgency coming on suddenly, with a desire to pass water at once. During the passage of water there is some scalding and a sense of relief for a time when passed. But afterwards there is a feeling of heat along the urethra, which gradually leads to another desire to pass water. The mucous membrane of the urethra is often swollen, and often there is urethritis and some degree of cystitis. *Phosphaturia* caused by the deposition of calcium phosphate in urine, alkaline from the presence of some fixed alkali, whether this body is in excess or simply deposited from the alkaline urine, gives rise to rather a characteristic dysuria. The calcium phosphate, being a heavy salt,

¹ Des Dyspepsies, p. 144. Paris, 1857.

² Disease of the Stomach, p. 100, 3d edition, 1872.

deposits from the urine, as it collects in the bladder, at the base, so that the first portion passed is either clear or milky and passed without much irritation, whilst towards the end the accumulated calcium phosphate passes as a creamy discharge with much straining and irritation. To this the late Mr. Marcus Beck happily applied the term "terminal" phosphates. Another symptom of dysuria that frequently denotes this form of phosphaturia is an aching pain just above the pubis. The phosphaturia arising from ammoniacal decomposition of the urine also causes the most intense dysuria, but as it is generally associated with disease of the genito-urinary tract, we will consider it more fully under that head.

Oxalate of lime, when present, often mixed with uratic and phosphatic deposits, certainly aggravates the symptoms. It is, however, frequently the sole abnormal deposit present except mucus, and the dysuria of oxalate of lime seems especially to affect the prostatic portion of the urethra. Whilst uric acid or the urates cause a general heavy burning pain along the course of the urethra, and phosphaturia causes pain at the neck of the bladder, especially noticeable at the end of micturition, patients with oxaluria complain of a burning pain in the region of the perineum, radiating upward towards the rectum. Micturition gives little or no relief, for as soon as the bladder is emptied the burning pain commences again.

Sugar may be present for a considerable time in the urine without giving rise to any pronounced dysuria except that caused by undue frequency of making water, owing to the diuresis. But after long continuance the sugar causes irritation of the urinary passages, especially round the meatus of the urethra, and in time may induce cystitis.

Albumen, Blood, Pus.—The mere presence of these products in the urine causes little or no irritation in their passage. Often a considerable quantity of albumen may be found in the urine without any attention being drawn to an abnormal state of micturition. When such is caused it is generally in connection with active renal trouble (reflex). The same may be said with regard to pus and blood; when dysuria is occasioned by these it is either owing to the passage of stringy mucopurulent masses, or coagula; or from reflex irritation. With regard to this latter condition, I have met with symptoms closely resembling those of vesical calculus in two patients suffering from hæmoglobinuria, i.e., great urethral irritation, pain at the end of the penis always preceding the discharge of a little blood and some coloring matter, although the urine itself was free from other irritating products except albumen and some free uric acid. I cannot find that this dysuria

has been noticed in relation to hæmoglobinuria before. It is important, as it may mislead us into a diagnosis of stone; in fact, both the cases I allude to were sent to me under that supposition. The paroxysmal character of the dysuria and its complete disappearance between the attacks ought to help us to a right conclusion, which would be confirmed by a microscopic examination of the urine during a paroxysm.

Poisons.—Cantharides and turpentine are the two which for practical purposes we need only refer to, as their well-known effects of producing extreme dysuria are likely to occur to us in our clinical experience. The extreme susceptibility of some persons to cantharides is well established, the mere application of a small blister of Spanish fly causing very severe dysuria and bloody urine. Such an extreme susceptibility has not been established with turpentine, which is sometimes administered in large doses. But some time ago I was summoned to see an elderly lady who was supposed to be suffering from an acute attack of renal colic. There was extreme dysuria with very scanty bloody urine and considerable pain in the lumbar region and vomiting. At first I was misled, but on inquiry I found that she had previously applied a turpentine fomentation to her chest for some bronchial trouble, and as its toxic effect passed off no symptoms of renal calculus persisted; I could, therefore, only conclude that this violent attack of dysuria was due to the small quantity of turpentine that had been absorbed by the skin.

II. MORBID CHANGES IN THE GENITO-URINARY TRACT.

These, as already stated, come chiefly under the observation of the surgeon in connection with stricture of the urethra, enlarged prostate, stone, etc., with which the scope of our remarks does not permit us to deal. Simple *idiopathic cystitis*, however, is a condition which frequently calls for attention from the physician, either as limited to the bladder, or else the inflammation extends through the whole length of the genito-urinary tract. After long-continued debilitating illness cystitis often appears, generally associated with an alkaline state of the urine and the deposit of calcium phosphate, which appears to be the exciting cause. Elderly persons also appear to be more susceptible to catarrh of the bladder than those of middle life or younger years. Some, too, of all ages seem more susceptible to vesical catarrh at all periods of life than others; just as some persons are more troubled with bronchial catarrh, and others with catarrh of the gastro-intestinal tract, without our being able to assign any special cause. But above

all the causes of cystitis that come under the observation of the physician, that following lesions of the spinal cord is the most frequent. The symptoms are the most marked because there is a special tendency to ammoniacal decomposition and the formation of crystals of ammonium-magnesium phosphate (triple phosphate) which greatly aggravate the dysuria. It is an open question whether such decomposition can be set up in the bladder without the introduction of the specific organism by means of the catheter, as some maintain. Others hold that the peculiar secretion from the mucous membrane of the bladder furnishes the necessary ferment and culture medium. Certainly one meets with cases in which ammoniacal urine does occur without there being any evidence of the cause having been introduced from without. This may be owing to imperfect observation, for the urines of patients suffering from spinal diseases, even when passed fresh and acid, rapidly undergo ammoniacal decomposition. I have observed urine clear when passed, with an acid reaction, becoming ammoniacal and depositing crystals of triple phosphate before the urine had become cool. In addition to the irritating qualities of the ammoniacal urine and crystals of triple phosphate, the dysuria is greatly aggravated by the stringy masses of muco-pus that collect and form in the bladder. Another form of cystitis which particularly affects the aged, *dysuria senilis* (Harnscharfe der Greise), comes on very insidiously and frequently claims the attention of the physician. The complaint is usually attended with some paralytic condition of the lower bowel, constipation being always a marked initial symptom. The skin becomes harsh and dry; the tongue covered with a white fur, but red at the edges. At the same time severe pain is often experienced in the thighs and legs, whilst a papular itchy eruption (lichen) is constantly present, affecting chiefly the abdomen and inner surfaces of the thighs. The quantity of urine becomes less and less and higher in color till it assumes a deep brown. No morbid constituents are usually present: it seems merely concentrated urine. Micturition is attended with much pain, and the fluid excreted is so irritating that the skin of the prepuce and insides of the thighs becomes excoriated by contact. It has also such a high urinous odor that, though the greatest care may be taken to prevent contact with the under linen, the patient has always a urinous odor. These cases may run a protracted course, but frequently they usher in a serious train of symptoms that end fatally in coma. In two cases that have been recently under my observation, one a male aged seventy-five, the disease began with obstinate constipation and with the gradual diminution of the urinary secretion, until

the patient died in coma three weeks after the first symptoms showed themselves. The other was a female aged seventy, and in her case the symptoms have been more prolonged. She has gradually emaciated and has suffered from constant dysuria both night and day. The urine is simply concentrated and highly charged with urates. There is thirst and intolerable itching of the skin. No explanation of this condition has yet been offered.

Another form of *dysuria senilis* comes on more insidiously ; indeed, it often exists before the patient becomes aware that anything is particularly the matter with the urinary tract. In these cases two conditions seem to bring it about,—(a) an atonic state of the muscular coat, and (b) diminished sensibility of the mucous surface. Incontinence of urine and retention here play a double part. The bladder is always full and the slightest motion of the body causes the urine to escape. The consequence to which such a distended state of the bladder leads is that the cavity becomes enormously enlarged, and the power of emptying it naturally becomes less and less. The amount, therefore, of residual urine is consequently constantly on the increase, whilst the sensibility of the mucous surfaces, which before from abnormal apathy failed to warn the patient the bladder was full, now becomes extremely irritable, causing on the passage of the smallest quantity of urine severe and prolonged pain. At the same time the effects of the back pressure upon the ureters and kidneys begin to manifest themselves. The patient now becomes extremely restless, worn out with the frequent efforts at urination, and speedily falls into a uræmic condition.

Gouty cystitis, or, to speak more accurately, cystitis occurring in a gouty patient, often gives rise to a troublesome inflammation of the bladder, the more so since the cause is often overlooked, or rather the diseased condition is attributed to some other cause. The symptoms come on very insidiously, increased frequency of micturition, especially at night, leading to a suspicion of nephritis. Nothing abnormal is, however, to be found in the urine, except pus, with the occasional discharge of urates. But these are not constant, nor has the urine any very high degree of acidity. In spite of the prolonged pyuria the patients apparently do not suffer in health, indeed are often better whilst the urine remains purulent. Like many other gouty manifestations, it is characterized by its tendency to rapidly disappear and reappear without apparent cause. In some cases of apparently gouty origin the mucous membrane of the bladder exfoliates in shreddy masses sometimes, though more rarely in its entirety (*cystitis exfoliativa*). Closely allied to gouty cystitis is *gouty urethritis*, often appear-

ing at the same time, but usually preceding extension to the bladder. It is well to caution you in regard to the appearance of this urethral discharge, which very often may attain a high degree of severity in middle-aged adults, as I have known physicians give unpardonable offence by attributing its presence to a wrong cause. Diabetic patients too frequently suffer from urethritis and consequent cystitis. In these cases I have observed that the irritation does not usually originate in the mucous surface of the genito-urinary tract, but that it creeps backward from the external surfaces, the prepuce, the vulva, labia, etc., which have become the seat of eczema. Indeed it is doubtful whether the passage of sugar can excite inflammation of the internal mucous surfaces of the genito-urinary tract. When, however, cystitis does occur in the course of diabetes, however excited, it adds considerably to the patient's trouble, and sometimes is attended with the phenomenon known as *pneumaturia*; the saccharine element undergoing vinous fermentation in the bladder, and disengaging carbonic-acid gas, which causes painful distention of that organ. A very insidious form of *urethritis* I have occasionally met with in delicate women, which had escaped observation and had given rise to much distress. In one case a young lady aged twenty-nine had suffered for six years from a most distressing dysuria, and had consulted numerous special authorities without relief. Several had confidently told her it was hysteria. The urine on examination was normal except for an excess of mucus and a few pus-corpuscles. A surgical examination proved there was no lesion of the bladder, and an examination per vaginam proved the absence of any pelvic disease. She was relieved by drinking three ounces of distilled water every two hours during the day; but the persistence of a few pus-corpuscles in the urine showed there was irritation somewhere in the course of the genito-urinary tract. This after some time was discovered to proceed from the urethra, which upon a more exact examination was found hard and cord-like when felt per vaginam, though no apparent exudation of pus could be observed from the meatus. Injections of nitrate of silver at length cured her. It may be mentioned, however, that whilst she was on a course of Contrexeville water she discharged large quantities of uric acid, nearly a teaspoonful at a time, so that probably some part of her trouble was due to reflex irritation. I have since seen cases resembling this, in respect to the dysuria, and it should always be borne in mind when females complaining of incontinence of urine, which may have been hastily referred to neurosis, come before us. A *granular condition* of the meatus in females frequently gives rise to a very troublesome dysuria. You will most of

you remember a case in No. 10 bed (Charlotte ward) of a middle-aged woman, in whom this symptom failed to yield to treatment until an examination of the external parts informed us of the condition, after which it rapidly yielded to the application of silver nitrate. Like granular conjunctivitis, it seems particularly to affect persons of full habit and gouty proclivities.

Tubercular cystitis hardly comes within the scope of our consideration to-day, but I need hardly say that no form of bladder disease is attended with more constant symptoms of pain, hemorrhage, and purulent discharge; this added to the constitutional disturbance generally renders the diagnosis easy.

III. DISTURBANCES OF INNERVATION.

These are generally reflex. Take for example *stone in the kidney*, which is often attended with symptoms that point to the presence of calculus in the bladder. One patient, in whom Dr. Prout had diagnosed stone in the kidney forty years previously, experienced such attacks of dysuria that nothing could persuade her that she had not a vesical calculus, in spite of repeated examinations by such experts as Sir William Ferguson, Sir Henry Thompson, and Sir Prescott Hewitt. The renal calculus, which had attained an enormous size, could easily be felt in the left kidney, and the attacks of paroxysmal dysuria were evidently caused by a collection of pus distending the pelvis of the kidney; when this came away, as it did after a time with a sudden gush, relief was immediately obtained. I need hardly remind you of the dysuria that attends *acute nephritis*, and the nocturnal frequency of micturition associated with *chronic Bright's disease*. Reflex dysuria follows upon any engorgement of the lower bowel, as, for example, the *tenesmus* of dysentery, or internal hemorrhoids. There is, moreover, a dysuria caused by general plethora of the pelvic vessels which may escape attention unless there is also evidence of internal piles. Sometimes instead of hemorrhage from the bowel free bleeding takes place from the prostatic vessels, giving rise to serious alarm and to erroneous diagnosis. I know a patient of full habit who for many years past has bled freely twice a year or oftener through the bladder, and has not exhibited any symptom of piles, and which is no doubt caused by fullness of the prostatic vessels, and who still retains his general health. The hæmaturia is usually preceded by a feeling of weight in the perineal region and pricking pains up the rectum. Since we have been able to dismiss from our minds the fear that he was imminently in danger of sinking from villous growth in the urinary organs, we have

regarded it as a wholesome effort, the prostatic veins bleeding instead of the rectal.

Spasm of bladder, so frequently mentioned by the older writers, is usually associated with some of the previously described causes of dysuria, principally oxaluria; indeed, I have hardly met with a case in which oxalates were not abundantly present in the urine. The symptoms come on usually in the night-time; first in colicky pains above the pubes, then a violent inclination to pass water, but this only comes in small quantities with forcing pains shooting back from the bulb of the urethra and involving the sphincters and levatores ani in a general spasm. It seems to be brought about by a general reflex irritation caused by the elimination of irritating products. In the cases I have been called to see, the attack usually followed some indiscretion of diet. Dysuria due to reflex irritation in young children may be brought about by very slight causes, the chief of which may be referred to *intestinal worms*, or the irritation of an *elongated foreskin*. Retained uratic infarcts in the kidney, as well as formed calculi, naturally cause considerable reflex irritation. With regard to the direct disturbances of innervation, we are only aware of those that affect the sensibility of the mucous membrane and trophic changes in the muscular wall of the bladder. Of these, the foremost are found in the so-called "*urinary crises*" of locomotor ataxy as examples of excited sensibility; and in the involuntary incontinence of young children, who will often wet themselves in the daytime as well as at night, in spite of the most careful supervision, as pointing to a state of anæsthesia of the urinary tract in which no warning is sent to the micturating centre. Lesions of the spinal cord such as produce paraplegia are followed by trophic changes, and, as Owen Rees pointed out, are at first attended by a glairy discharge of acid mucus; this on the introduction or development of the specific micrococcus becomes specially the seat of ammoniacal fermentation. This primary fermentation in a urine of acid reaction, with precipitated masses of stringy mucus, I have repeatedly observed in the earlier stages of acute spinal lesion, thus confirming Dr. Rees's observation. The rapid change to ammoniacal fermentation cannot, however, always be attributed to catheterism, and a sufficient cause has still to be alleged for its sudden and often unaccountable supervention. It may be that the ammoniacal ferment is always more or less present in the urinary tract, but that only under certain conditions does it find opportunities for development.

Such, gentlemen, are the chief causes of dysuria that come under the special notice of the physician, apart from those more serious ones that

engage the attention of the surgeon or the gynæcologist. It will be readily seen that our measures of relief, if they are to be successful, must depend on our right determination of the conditions that produce them. To take the more simple forms, those depending on alterations of the quality of the urine. For simple acidity of the urine, with or without uric acid or uratic deposits, the mere directing the patient to drink three ounces of distilled water every two hours is often sufficient. In many cases, especially of young children who are specially affected with this condition, it may be advisable to limit the nitrogenous diet, replacing it with milk and farinaceous food. In phosphaturia consequent on alkaline urine (fixed alkali), the administration of dilute hydrochloric acid with *nux vomica* is often sufficient. But in the more severe cases attended with wasting and general disturbance of metabolism, some of the milder preparations of opium are decidedly of advantage. This is specially the case if the tongue is furred in the centre and red at its edges. Dilute nitro-muriatic acid is always beneficial if oxalates are found in the urinary deposit, whilst a meat diet should be adopted to the exclusion of all saccharine and farinaceous articles of food.

Acute catarrh of the bladder, whether idiopathic or consequent on pre-existing disease, is often attended with great dysuria and febrile reaction. In urgent cases great relief usually follows the application of leeches to the perineum; and if there is much febrile disturbance, five-grain doses of *pulvis antimonialis*, combined with an equal quantity of Dover's powder, are generally followed by a remission of the more urgent symptoms. In milder cases, especially with an acid reaction of urine, twenty drops of *liquor potassæ* with forty to sixty drops of tincture of henbane, given every four hours, are sufficient to abate the urgent dysuria. It is interesting to observe the efficiency of henbane as a sedative for all painful affections of the urinary tract, since it has only a feeble effect on the bronchial mucous surface, and apparently none on the gastro-intestinal. Linseed poultices over the hypogastric region and between the thighs should not be forgotten. As a minor matter of relief, the patient should be directed to sit over a vessel filled with hot water (*bidet*), or the chamber vessel should be half filled with hot water whenever the attempts to micturate come on. If full doses of henbane do not relieve the dysuria, then we must fall back on ten-grain doses of Dover's powder given two or three times a day. When the cystitis becomes chronic, if the urine is still acid, great benefit will follow the administration of the benzoates; and for this purpose I prefer the use of ammonium to the lithium salt, as the former is more di-

uretic in its action. Combined with this may be given strong infusions of buchu, uva ursi, etc. Any weakness that may follow on any prolonged attack, and also to guard against relapse, will be best met with the prolonged use of Contrexeville water and balsamic remedies, of which the best is "sandal-wood oil" given in the form of capsules. When much atony is left, a mixture of iron with strychnine is decidedly beneficial. In the cystitis attended with alkaline (ammoniacal) urine we must not overlook the fact that in the first stage the urine may have been not only acid, but highly acid, and that alkaline reaction has only been caused by the accidental introduction of a micro-organism. For this reason Dr. Owen Rees advocated the administration of liquor potassæ to neutralize the effect of the formation of the acid mucus which he believed was secreted from the bladder in spite of the ammoniacal fermentation taking place in the urine. Whatever good may be obtained by this remedy in certain stages of the disorder, I cannot sanction it as a general practice, and it should not be attempted till the activity of the ammoniacal fermentation has been considerably lessened. Then, and not till then, I have often been gratified by noticing the improvement in the patient's condition following its administration. Unlike the cystitis met with when the urine is acid, ammoniacal cystitis is more insidious in its onset, less violent in its progress, but infinitely more difficult to eradicate and dangerous in its results. Whenever it is possible, and this is more a question for the surgeon than the physician, the fermentation in the bladder should be dealt with directly by means of injections. In simple cases, such as come under the observation of the physician, I usually direct the bladder to be irrigated with about two and a half pints of dilute citric acid (ten grains to one pint), and afterwards slowly fill the bladder with some disinfecting agent, of which I find eucalyptus, salol, or boracic acid the most effectual. This injection should be made as often as the catheter is used, —two, three, or more times a day. If catheterism is not required, then it is advisable only to use the injection at the time of washing out the bladder. Sir William Roberts, remarking in a case of glycosuria with ammoniacal urine that the casual introduction of some germs of lactic-acid fermentation substituted that reaction (acid) for the ammoniacal (alkaline), has proposed the injection of a solution of *bynin* (malt extract) in cases of ammoniacal cystitis to develop this opposite fermentation. Although the suggestion looks well on paper, it has not yet been tried clinically, and many objections might be raised with respect to its continued employment, and whether lactic-acid fermentation might not be as difficult to deal with as the ammoniacal form when established.

But it might be tried in severe forms and in cases where it was important to prevent the extension upward from the bladder, along the ureters, to the kidneys themselves. Of internal remedies modern research has placed two in our hands which enable us to deal on more equal terms with this obstinate condition. With *salol*, if there are no contra-indications in the form of renal disease, we may safely affirm that we can arrest ammoniacal fermentation of the urine after a few doses; and in time, no doubt, aided by topical applications to the bladder, can prevent its recurrence. I have seen a highly ammoniacal urine that had previously resisted all forms of treatment become acid in reaction, and, with the exception of some pus, otherwise normal within three days of commencing the drug, in doses of twenty grains three times a day. Unfortunately it cannot be used with safety if the kidneys are degenerated; here, however, we may fall back on boracic acid, which certainly often has a highly beneficial effect, though for certainty it cannot be compared with *salol*. This last I have found useful in cases of cystitis in diabetic patients, especially in those cases in whom a certain degree of pneumaturia occurs. The dysuria arising from *gouty cystitis*, or urethritis, must be treated on the same lines as that caused by excessive acidity,—viz., the ingestion of three or more pints daily of soft water. Its use should be continued in more moderate potions after recovery, to prevent a relapse. The diet should be only slightly nitrogenous and the bowels carefully regulated. The somewhat persistent pyuria which attends this condition is best treated with injections of silver nitrate. It is probable, as in *gouty conjunctivitis*, that the mucous membrane of the bladder becomes the seat of excessive purulent secretion without any great degree of hyperæmia. In the chronic urethritis of females injections of nitrate of silver alone proved serviceable, though even then the complaint was very troublesome to eradicate, owing to the frequent relapses and to the thickened condition of the mucous tissues from past neglect.

Spasm of the bladder, the result of reflex disturbance, can only be effectually dealt with by one drug,—opium. "Thank God for opium," wrote John Hunter when referring to this subject, and the dictum cannot be improved, even in these days, when more fashionable hypnotics have made us less familiar with its virtues. It should be given in all forms of reflex vesical irritation. Even in Bright's disease it may be given, if the dose is carefully adjusted, to allay the often troublesome nocturnal micturition. In these cases the lozenges of morphia (one-thirty-sixth grain) with ipecacuanha, of the British Pharmacopœia, are very serviceable, as providing us with a safe dose of morphia and

permitting of its gradual increase within limits of safety. With regard to *dysuria senilis*, there appears to be a failure not only of the vesical muscles, but also of innervation, whilst in the acute form at least there appears to be a failure of the aqueous secretion of the urine. For the treatment of this condition little can be done, but I have noticed some temporary benefit follow twenty-drop doses of dilute nitric acid; why, I do not know, but apparently by increasing the quantity of urine and rendering it less concentrated. In the chronic form our success is usually more gratifying. Here the best results follow on complete and systematic catheterism. The bladder should never be allowed to become distended, however frequently the catheter has to be employed. As soon as the cystitis, that may have been caused by the presence of residual urine, has been dealt with, we should begin with perchloride of iron, strychnine, and belladonna. If treatment has not been delayed too long, till back pressure has also caused dilatation of the ureters and kidneys, these patients often regain considerable control over their bladder, and may be able, for a time at least, to do without the frequent passage of the catheter. Massage over the hypochondriac region and use of the faradic current are also powerful adjuncts.

The question of incontinence in children is too wide a subject to be treated in the time allotted for our lecture to-day, but there is one form to which I have already alluded, in which the patient involuntarily passes water during the daytime when awake, as well as in the night-time when asleep, and apparently does not know whether the bladder is full or empty. These cases are even more obstinate than other forms of juvenile incontinence; those may yield to ergot, valerian, etc., but this hardly ever shows any beneficial result from the use of drugs. The only improvement that may be considered at all obvious is in a case at present under observation in Charlotte ward, which, after the persistent use of all kinds of drugs, has shown a degree of amelioration from the daily application of the faradic current over the hypogastric and lumbar regions.

URETHRAL FISTULÆ.

CLINICAL LECTURE DELIVERED AT THE COLLEGE OF PHYSICIANS AND SURGEONS.

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GENTLEMEN,—The case which I present to you this morning is one of considerable interest. The patient, as you see, is a man of middle age. He has suffered for over ten years from a deep urethral stricture. This has been treated in a desultory fashion, and he has had several attacks of retention prior to his consulting me. I found on examination that the stricture was impermeable, excepting to a filiform bougie, and that there were numerous false passages present, which had been produced by violent attempts at instrumentation with small instruments. I attempted treatment of the stricture by dilatation, but after some three weeks' careful attention, I found that not only did my treatment give rise to urinary fever, but the stricture proved very irritable and resilient, and was absolutely rebellious to dilatation. Under these circumstances, the only recourse was perineal section, which operation was performed about three months since. As the stricture was multiple and extended well forward to the base of the scrotum, the perineal section extended farther forward than is customary in such cases. Healing was by no means satisfactory; and, as you see, the process of repair was incomplete anteriorly at the base of the scrotum, as a result of which a permanent fistula remained. This has been treated in various ways without success. I have, therefore, decided to do a plastic operation this morning for the purpose of closing the fistula.

Apropos of the case in hand, it might be well to discuss in a general way some of the points involved in connection with the subject of urinary fistulæ. Urethral fistulæ, as a rule, result from extravasation of urine and peri-urethral abscess. They may, however, result from traumatism, accidental or deliberate, as in this case. The fistulæ are usually situated in the vicinity of the perineum and scrotum, but cases have

been observed in which numerous fistulæ opened in the groin, the inner side of the thigh, or on the wall of the abdomen. These erratic locations of fistulæ are due to the extensive burrowing of pus. The point of departure of urinary fistulæ is usually found somewhere in the neighborhood of the bulbo-membranous region of the urethra. Occasionally they are met with in and about the scrotum, or in the pendulous portion of the urethra. Perineal urinary fistulæ are very often multiple, numerous openings being found about the scrotum, nates, perineum, or inner side of the thighs. Civiale reports a case in which there were, I believe, more than fifty external orifices of fistulæ, the point of departure of which was the perineal portion of the urethra. As is true of fistula in ano, the pus in cases of this kind may burrow about in a most erratic manner. The importance of fistulæ varies according to whether they are single or multiple, their situation, diameter, and length. It is to be noted that perineal fistulæ, having one or perhaps two orifices, are not, as a rule, of very great importance. They will usually heal spontaneously, provided all obstruction of the urethra has been removed. I recall a case of my own in which a very narrow meatus coexisted with a deep perineal stricture and perineal fistula. Operation upon the meatus effectually cured the fistula. The size and importance of fistulæ depend to a great extent upon the amount of substance of the urethral wall which has been lost. The orifice of the fistulæ may be large enough to admit several fingers. As a rule, they are irregular and of considerable depth. The external orifice may be quite narrow, and healing may occur from time to time with a resultant accumulation of the pus in the fistulæ. This pus forms a distinct abscess which subsequently ruptures, leaving the fistulæ in worse condition than ever. When the stricture is of very small calibre, and a fistula is associated with it which is comparatively of large size, the urine may not escape through the normal channel, but instead entirely escape through the fistula. It is very rare that a stricture becomes completely agglutinated, although such a misfortune is quite likely to occur in cases of traumatic stricture with complete division of the urethra.

The treatment of fistulæ depends upon the circumstances of location, number, and the question of the loss of substance or otherwise. The first principle in the management of fistulæ of the urinary tract lies in relieving all obstructions to the outflow of urine. A contracted meatus and strictures in the penile portion of the canal require division by the urethrotome. In removing obstructions of the urethra in cases of urinary fistula, great caution should be observed to completely restore the calibre of the canal, for the more perfectly this is done the

less friction or resistance is there to the outflow of urine. By this procedure we take advantage of the well-known physical principle that fluid has a tendency to flow in the direction of least resistance. Should there happen to be, at any part of the urethra, a sufficient degree of obstruction to distend the urethra behind the obstruction, the backward and outward pressure will necessarily force a portion of the urine into the internal orifice of the fistula, and thus prevent healing. It will be found, however, that the majority of simple perineal fistulæ will close spontaneously just as quickly as the normal calibre of the urethra has been restored. The reason for this spontaneous healing in the perineal portion of the canal is simply because the tissues in this situation are thick; and, as a consequence, reparative action is much more active than in the rest of the canal. Again, the parts are not disturbed by sexual excitement. Thus they have an opportunity to rest,—a very important matter, inasmuch as any disturbance of circulation necessarily interferes with granulation and retards the healing process. In spite of a thorough operation for the relief of urinary obstruction, urethral fistulæ do not always close spontaneously, and it is necessary to adopt some measure to stimulate the process of repair and bring about cicatrization of the fistulous tract. The main factor in perpetuating fistulæ is necessarily the entrance of urine into the tract of the fistula. This may, to a certain extent, be obviated by the regular use of the catheter in some cases. In some instances, the *sonde a demeure*, i.e., the retained catheter, is effectual. Simple cauterization with the nitrate of silver, tincture of iodine, or nitric acid is oftentimes effectual. When these means fail, as they are very likely to do if there is much loss of substance or if the fistula is penile, it is necessary to do a plastic operation for the closure of the fistulous tract. Two points must be considered in doing a plastic operation upon the urethral canal: First, to get a good covering of new and healthy tissue for the fistulous opening. Second, to prevent the urine from coming in contact with the freshened surfaces. Numerous plastic operations have been devised to fulfil the first indication. As far as the second indication is concerned, it is really amusing to note the agonizing efforts which some surgeons make to keep the urine from coming in contact with the field of operation. The retained catheter, or the practice of interrupted catheterization, is most generally resorted to. Infection is almost sure to occur with the retained catheter, to say nothing of the mechanical irritation induced by the constant presence of a foreign body in the urethra. Frequent catheterization, on the other hand, produces disturbance of the wound, with the result of interfering with the process of repair. There are some

cases in which it is impossible to avoid keeping a sound or catheter in the urethra for some days, or perhaps weeks, after the plastic operation ; but this is of comparatively little moment, providing suitable means be employed to divert the urine from the wounded area. It is to be remembered that a simple perineal puncture of the membranous urethra, with drainage of the bladder through the perineum, is not only a simple operation, but is in most instances absolutely safe. By the perineal puncture and drainage we may at once set aside one of the principal obstacles to success of plastic operations upon the urethra,—namely, contact with, and mechanical disturbance by, the outflowing urine. Simply paring the edges of the fistula and subsequently stitching them together is very often successful, providing the urine be diverted from its ordinary channel ; but in very many instances the simple varieties of plastic operations, by virtue of the sparsity of the tissue, are not successful. In the penile portion of the canal it is especially difficult to perform a successful operation of urethroplasty. This fact results from the relative thinness and looseness of the skin and the scarcity of cellular tissue in this location. The tissues are so scanty in amount that the surgeon is naturally indisposed to pare the surfaces of the fistula to any great extent, and the paring process is consequently often insufficient to obtain the desired result. It is evident that thick-edged flaps, such as can be secured in the deep urethra,—i.e., the perineal portion,—unite much more satisfactorily than relatively thin ones, upon which we must depend in the pendulous portion of the urethra. Physiological variations in size are frequent in the penile portion of the canal, and constitute another very important source of failure in urethroplasty, inasmuch as these variations in size produce a certain degree of tension of the tissues, and necessarily preclude the possibility of the proper amount of rest.

One of the best operations of urethroplasty, and the one which I am about to perform in the case before us, is that of Szymanowski. This operation is performed in the following manner: When the fistula lies in the long axis of the penis, a straight incision is to be made, beginning just in front of the fistula and terminating a short distance behind it. The integument upon one side is then to be dissected up so as to be freely movable. A half-oval flap of skin on the other side of the fistula is then outlined and dissected up, excepting at the edge of the fistula, its epidermis being first removed. The dissected flap is then to be inverted and pushed under the skin, which has been freed upon the opposite side, as into a pocket. It is then to be retained in position by sutures passed into and through the bottom of the pocket.

The movable skin is then slid over it and also stitched. An elastic catheter is to be passed into the bladder and there retained. This operation has been practised and highly recommended, especially by such excellent operators as Drs. Charles McBurney, Robert F. Weir, E. L. Keyes, and T. T. Sabine. I have employed the method in two cases with most gratifying success.

Dr. McBurney has practised a modification of the operation in a number of cases with great success. In a report of six cases this gentleman speaks as follows :

One case, the first, owing to imperfect management, failed completely. Five cases succeeded, and in none of them was any second operation required.

The second case required three months and six days to obtain sound healing ; the third, seventeen days ; the fourth, thirteen days ; the fifth, nineteen days ; and the sixth, thirty-four days. No symptom of the slightest importance occurred in any of the last five cases, and the length of time occupied in recovery certainly compares very favorably with that often spent in futile and even successful attempts to close perineal fistulæ without operation.

The plan adopted in all of these cases was as follows ; and, if the description seems tedious, my excuse must be that I believe the success of the operation to depend largely upon a close attention to details.

Some time previous to operation the entire urethra was cleared of all evidence of stricture, and the urethra accustomed to the passage of sounds ; any sinuses in the neighborhood of the fistula were opened and soundly healed. If cystitis existed it was removed as far as possible, although a moderate amount of chronic cystitis certainly does not contra-indicate the operation.

The day before operation the bowels and rectum were thoroughly cleared, in order, especially, that for several days afterwards the rectum and adjacent parts might be kept perfectly quiet by a free use of opium. The perineum was then shaved and carefully cleansed, and the bladder emptied with the catheter, and thoroughly washed out with a weak solution of either borax or carbolic acid. The edges of the fistula were then either scraped or cut, so as to remove all suppurating granulations which would naturally increase discharge and prevent early union.

A single straight incision was then made from A, three-quarters of an inch in front of, to B, three-quarters of an inch behind, the fistula. (Fig. 1.) This incision passed through skin and superficial fascia, and closely skirted the right side of the fistula. The edge of this incision was raised, and, working with a small blade to the patient's right side,

the skin and fascia were undermined until a pocket was formed including the area A, C, B, F, the right edge of the pocket being indicated by the dotted line A, C, B.

On the opposite side a curved incision, A, D, B, was then made,

FIG. 1.

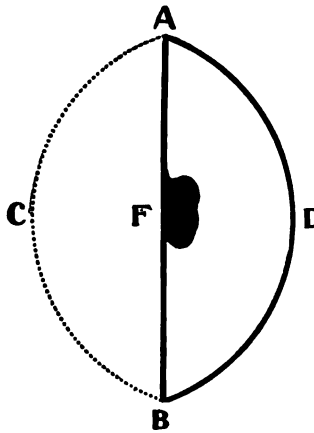


FIG. 2.

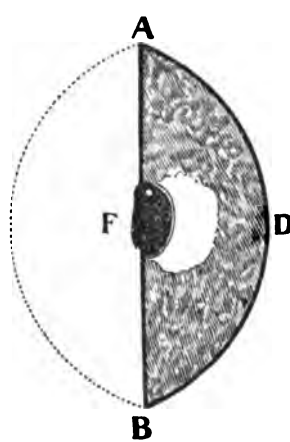


FIG. 3.

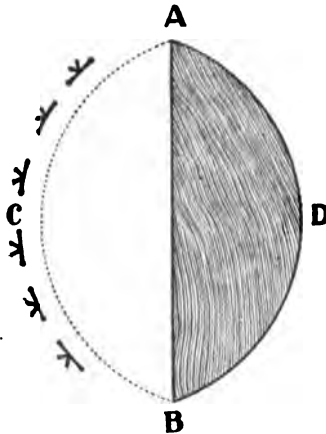
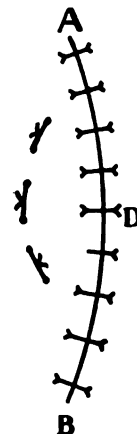


FIG. 4.



the greatest width of the flap thus marked out being three-quarters of an inch to one inch.

This flap must be generous and should include a good padding of fascia, as, when it is lifted, the shrinkage is great.

Before lifting the flap, a thin layer of skin was removed from its surface. This is best done with small, curved scissors, the superficial layer of skin being rapidly chipped off.

The freshening process was carefully extended over the entire area A, D, B, F, excepting over a surface a little larger than the fistula, and immediately next to it.

It was thought best to leave this portion undenuded for the immediate cover to the fistula, because less cicatricial repair would occur and less pus would be formed than if a raw surface were presented to the urethra. (See Fig. 2.) The flap A, D, B was then dissected up close to the median line and inverted, its attached edge acting as a hinge and as a medium for blood-supply.

Five or six fine catgut sutures were passed through the skin at different points a little beyond the dotted line, A, C, B, into the pocket, then through the free edge of the flap, and then back into the pocket and out through the skin. Five or six loops were thus formed, by drawing upon which the flap was closely drawn down to the bottom of the pocket, and the free ends of the loops were tied. (See Fig. 3.) Two or three sutures of catgut were now passed with a curved needle through the upper surface of the inverted flap so as to firmly bind it to the parts beneath. Sometimes with interrupted and sometimes with a continuous catgut suture the free edge A, F, B was now securely fastened to the edge A, D, B. (See Fig. 4.) Irrigation with carbolic acid or bichloride solution was used throughout, excepting in the first case.

The dressing consisted of iodoform, iodoform gauze, and a cotton pad, held in place with a T-bandage. A morphine suppository was usually introduced before the dressing.

The subsequent treatment consisted in a free use of opium to prevent the rectum from acting, and the use of the soft catheter; the latter at least every six hours, and as much oftener as was required. Sometimes the catheter would be required as frequently as every three hours, and sometimes it caused moderate urethritis.

The catheter was always, excepting in the first case, thus used: it was introduced and the water drawn off. The bladder was then gently washed out with a weak solution of either carbolic acid or borax; I prefer the latter. On withdrawing the instrument the end was tightly pinched until the whole catheter had been removed from the urethra. This plan seemed to reduce the chance of contaminating the wound with urine from the inside to a minimum, and is certainly much to be preferred to the practice recommended by Szymanowski of tying in a catheter, or that made use of by me in my first case of puncturing the bladder through the rectum.

My own experience with Szymanowski's operation embraces in all

five cases, and the result has been in every instance satisfactory. Two of the cases were in the penile portion of the canal, and three were perineal fistulæ. I am free to say that I have little confidence in any operation for urinary fistula that does not involve the diversion of the course of the urine in such a way that it cannot possibly come in contact with the site of the plastic operation. In the two cases of penile stricture, I modified the operation to the extent of puncturing the membranous urethra through the perineum and drained by the perineal tube until the wound made by the plastic operation had perfectly healed. I believe that Szymanowski's operation will rarely fail in the penile urethra in moderate fistulæ, if this precaution be taken and the urethra be thoroughly asepticised before the plastic operation is performed. A solid bougie or moderate-size drainage-tube may be left in the anterior urethra, if desired. I used in one of my cases of penile fistula a rubber drainage catheter retained in the urethra, and I injected once daily into the urethra, through this tube, a mixture of iodoform and albolene to still further lessen the possibility of sepsis. In one case of perineal fistula, located so far forward that the base of the scrotum was involved, I found it necessary to use a flap of granulating tissue in covering in the fistula, and although the site of the operation was infected *ab initio*, I had a perfectly successful result. In some cases of perineal fistulæ located well forward, a comparatively simple operation has served me in several instances, instead of the more complicated plastic operations as illustrated by Szymanowski's method. You will find, in cases of perineal fistula following the ordinary operation of perineal section, that the defective union is almost invariably anteriorly. I have found in such cases that a free division of the perineum and the sublying urethra involving the fistula, followed by excision of the fistulous track down to the urethral wall, with subsequent stitching of the anterior portion of the wound, and drainage by a tube through the posterior angle, has usually been successful. I will state that in these perineal fistulæ one should not be in a hurry to perform a plastic operation of any kind, inasmuch as they may persist for some months, and yet finally heal completely. I have found, in some cases of perineal fistulæ following perineal section, that rest in bed for some weeks has proved perfectly successful without the employment of any further measures. We occasionally meet with cases in which simple catheterization will result in a cure of a small fistula in the perineum; but my experience is, as I have previously remarked, that both the retained catheter and intermittent catheterization tend to keep up irritation about the internal orifice of the fistula and favor sepsis, the result being a failure in by

far the majority of instances in which these measures are resorted to for the cure of the fistula.

In conclusion, I will state that the main indication for treatment in perineal fistulæ resulting from peri-urethral abscess, whether these fistulæ be single or multiple, is to remove the obstruction constituted by the stricture, which is usually the primary cause in such cases. Extensive and numerous perineal fistulæ can very often be cured by free incision of the perineal portion of the urethra, the knife being made to follow up all fistulous tracks, and, where it can be done without too much loss of tissue, the fistulous tracks being excised.

Gynæcology and Obstetrics.

PROLAPSE OF THE UTERUS—ALEXANDER'S OPERATION; ABDOMINAL SECTION FOR THE REMOVAL OF PAROVARIAN CYST.

CLINICAL LECTURE DELIVERED AT ST. LUKE'S HOSPITAL.

BY HENRY T. BYFORD, M.D.,

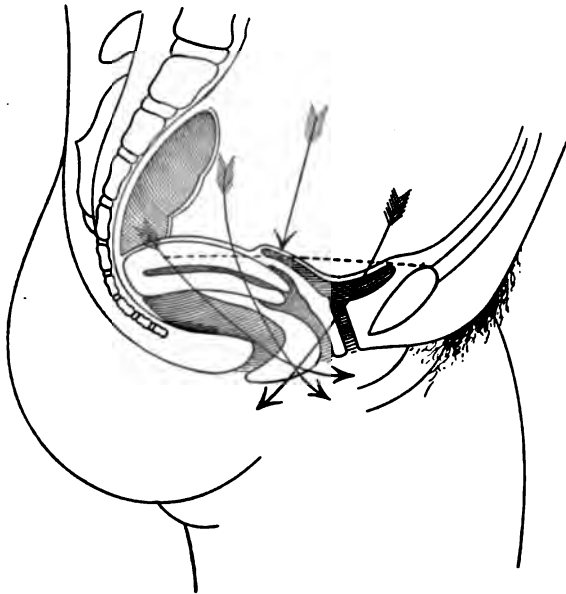
Professor of Gynæcology, College of Physicians and Surgeons; Professor of Gynæcology, Chicago Post-Graduate Medical School; Professor of Clinical Gynæcology, Woman's Medical College; Surgeon to the Woman's Hospital of Chicago; Consulting Gynæcologist to the Michael Reese, Charity, Chicago, and Provident Hospitals.

GENTLEMEN,—The patient now being anæsthetized is the one I operated upon before some of you three weeks ago. She then suffered from a procidentia uteri and a cystocele. The uterus was enlarged and the cervix eroded and lacerated. We curetted the uterus in order to remove the last traces of an old endometritis. I used a sharp curette and went over the endometrium quite thoroughly, but with extreme gentleness, in order to remove the membrane completely, but not to interfere with its cellular substructure and run the risk of preventing the reproduction of the mucous membrane in a healthier form. We then amputated the cervix after Schroeder's method, thus diminishing the weight of the uterus and promoting involution. The anterior vaginal wall was so extensively hypertrophied that we excised a large oval piece extending from near the urethral orifice almost to the cervix, and united the edges with transverse sutures of silkworm-gut. The strip was fully two inches wide at its centre. The perineum and relaxed vaginal entrance were denuded and sutured somewhat after the plan recommended by A. Martin, which is practically a modification of Freund's or Emmet's method, with extension of the lateral denudations in the sulci deep into the vagina.

While I am removing the stitches you can see that the parts are

pretty well closed up and look as if they would never relax again. But if I did nothing more, the procidentia would return in less than six months and there would seem to be as much redundant vagina as ever. The reason for this is that the uterus, although within the pelvis, is still prolapsed. The cervix is not much more than an inch from the vaginal outlet, a fact that enables me easily to remove the stitches through the small outlet. I have made two figures on the black-board to illustrate why the uterus will come out, and how we must prevent it. Fig. 1 shows you the uterus lying with its long axis corre-

FIG. 1.

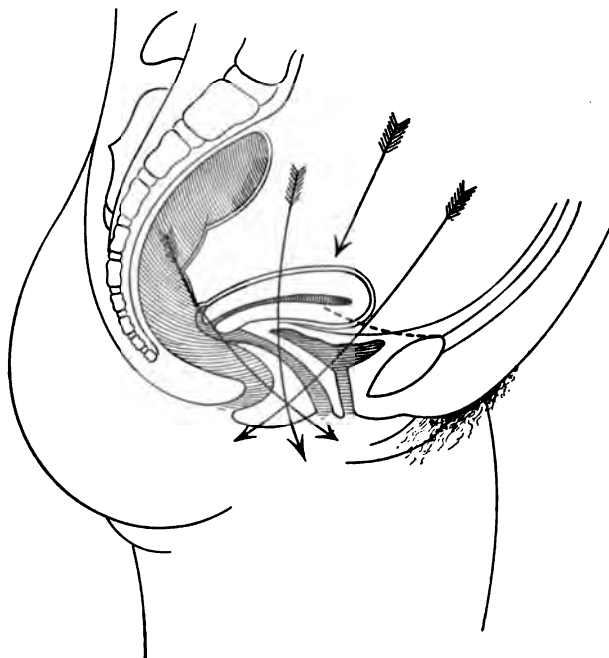


Position of prolapsed uterus as held by the plastic operation upon the vaginal outlet. Arrows indicate direction of abdominal pressure tending to force the cervix out at the vaginal outlet. The dotted line indicates position of over-stretched round ligament.

sponding with that of the vagina, having its smaller conical end in advance, ready with the aid of abdominal pressure, whose direction is indicated by the arrows, to pry, as it were, the anterior and posterior walls apart; or, more properly speaking, act as a wedge and force its way out. Now, we can prevent this if we can turn the uterus so that the long axis will occupy an acute angle with that of the vagina, as illustrated by Fig. 2. In this case the abdominal pressure forces the larger and longer anterior side of the uterus in the direction of the pelvic outlet. There is, of course, no chance for such a large surface to exert any pressure upon the vaginal outlet.

I will now ask my assistant, Dr. Walker, to perform the operation while I explain the steps. It matters but little at which side of the patient he stands, the most important consideration being to have a good light shining on the parts in order that the delicate tissues of the inguinal ring may be recognized before being cut. As the patient is thin, he easily finds the pubic spine and Poupart's ligament with the finger, and makes an incision about half an inch long over the spine and in the direction of this ligament. A finger placed in the cut easily palpates the external inguinal ring. The incision is now elongated to an

FIG. 2.



Position of prolapsed uterus after plastic operation and Alexander's operation. Arrows indicate direction of abdominal pressure, forcing the anterior wall of the uterus against the connective tissue barriers of the pelvic outlet. The dotted line indicates the position of the shortened round ligament.

inch in length. You will notice that there is scarcely any oozing of blood. In fleshy patients it is necessary to make a somewhat larger incision in order to admit more light. In such cases the external pubic artery on the inner side and the superficial epigastric on the outer side are apt to be cut.

The next important step is to cut down upon the inguinal ring without going through the intercolumnar fascia. If we cut through this

fascia rapidly, we are apt to incise the tissues within the canal, destroy the natural relation of the parts, and start up an annoying little hemorrhage. In checking this hemorrhage we disarrange the tissues still more, and may lose our ligament. Our operator is wise; he cuts down directly upon the pubic spine as an objective point and completes the dissection with the blunt-pointed fascia scissors, so that the deep fascia is exposed, and Poupart's ligament, the inguinal ring, and, in this emaciated patient, even the little soft elevation at the junction of Poupart's ligament and the spine where the round ligament is attached are plainly seen. The intercolumnar fascia covering the ring is now carefully incised in the direction of the inguinal canal, and the fascia surrounding the end of the round ligament is exposed. In this case I recognize the ligament immediately. We cannot recognize it in fleshy patients, but that does not make much difference, for we know that it stretches from the pubic spine for a short distance along the external column, or Poupart's ligament. Dr. Walker, as you see, takes up all of the tissue that lies against the external column in the grasp of the tissue-forceps, and therefore must have the ligament. A blunt hook is now pushed through the attenuated fascia under the tissues held by the forceps, and our ligament is securely caught.

Now comes the only difficult part of the operation,—viz., the separation of the ligament from its fascial connections so that it will slip or "run." We must snip or tear the delicate fascia that fastens the ligament to the sides of the canal. The secret of success in difficult cases consists in taking a fresh grasp with the forceps a little deeper in the canal each time the surrounding fibres are cut and the ligament gives a little. The operator is testing the ligament. He knows he has it, because one end of the tissues he holds runs directly to the pubic spine, and the other end gives an elastic resistance to traction. When edges or portions of the fascia are caught, they are inelastic and draw from the sides of the canal or abdominal wall. You notice the operator keeps hold of the ligament where it passes out of sight under the abdominal fascia. As he presses the connective tissue away from it, or snips a tiny band, it slips out a little, and he takes a fresh hold as far up as possible. The ligament is already pulled out nearly two inches, is increasing in size, and, as with his improved hold upon it he pulls a little harder, it commences to slip rapidly and easily, and is practically loosened. We do not now try to pull it out any farther, because the uterus is not replaced. The doctor is doing, as you see, exactly the same thing on the other side. Had the patient been a fleshy one and the ligament small, it would undoubtedly have been necessary to slit

up the inguinal canal for about a quarter of an inch in order to get a still better hold and a better view.

Now that the other ligament is also loosened, I replace the uterus with a sound and hold the fundus near the anterior abdominal wall over the pubes, while the operator pulls out the ligaments until they move the sound. Now that nearly six inches have been pulled out, I feel the sound drawn to one side by it, and tell him to stop for fear of breaking the ligament. Two slender silkworm-gut sutures are passed through the columns of the ring and the ligament, and after being tied are cut short at the knot. By means of a sponge a 1 to 2000 solution of bichloride of mercury is squeezed into the ring under the sutures and pressed out again. In cutting off the ligament enough is left to stitch along the bottom of the incision. The wound is closed with three deep and two superficial silkworm-gut sutures, the deep ones to be left one week and the superficial ones two weeks.

While the other side is being sutured, I will remind you that some prefer to open the peritoneal cavity and suture the uterus to the abdominal wall over the bladder. This I consider unnecessary in this case, because the plastic operations will provide sufficient support for the uterus as long as the fundus can be kept over the bladder. About the only objection to Alexander's operation is that the ligaments are in rare instances atrophied and of but little use. But even in that case no harm is done by such an attempt.

The operation should not be performed when there are pelvic peritoneal adhesions, nor when the uterus cannot be replaced so that the fundus will lie unsupported for a few seconds at least over the bladder, as a failure might result. A hysterorrhaphy would then be indicated.

I introduce a small Hodge pessary under the cervix and withdraw the probe. The patient will be catheterized or made to urinate every four or six hours, in order to prevent distention of the bladder. She will be kept in bed three weeks longer, and a daily douche will be given for four months. At the end of that time she may remove the pessary and consider herself cured.

The next case I present to you to-day is one of abdominal tumor. I have not made a thorough examination for the purpose of establishing the diagnosis, and I shall, therefore, proceed to do so in your presence. The patient is thirty-two years of age, married, and has been pregnant about ten times. She was infected by her husband with syphilis soon after marriage, and has had several successive miscarriages. The first child was born in 1881, but "died in three months of measles,"

it is said, but probably of syphilis. The second child was born in 1884,—still-born. She had a miscarriage in 1882, two in 1885, and another one in 1887, etc. With regard to her special symptoms, she has had more or less pain for two or three years across the lower abdomen. Last summer, about June or July, she first noticed an abdominal enlargement that has continued to grow since. There are almost no symptoms in connection with it, and our diagnosis will have to be made largely upon the physical examination.

Abdominal enlargements may come from various things. The most ordinary causes are meteorism, ascites, prolapse of the kidneys,—particularly of the right kidney,—ovarian and uterine enlargements, and various inflammatory changes. When there are any very decided symptoms in connection with these tumors, they may give some indications as to their nature. The absence of symptoms enables us to exclude malignant as well as tubercular disease, extra-uterine pregnancy, and hæmatoma. We may have prolapse of the kidney, ascites, omental, uterine, or ovarian tumor, or one of the various forms of tumors of the broad ligament.

I might say, while the patient is being prepared, that one of the commonest conditions you will find is prolapse of the right kidney. Every week or so I see a case or two of this kind in my office. The kidney is found under the ribs to the right of the median line, or very often occupying the space between the median line and the ends of the floating ribs. The chief signs are that the tumor is rather small, and can be pushed back into the kidney region almost beyond reach. Another important sign is elicited by putting the left hand under the ribs in the lumbar region and the right hand over the tumor, and then pressing into the lumbar region with the left hand so as to make the mass move against the upper or right hand. This suggests its retro-peritoneal character. Another diagnostic feature is that the dulness seldom extends under the border of the rib; there is intestinal resonance over the upper edges of it. In a case of enlargement of the liver the dulness is continuous with that of the liver region. Also in cases of enlargement of the liver you can often press the growth up under the ribs, but cannot displace it from them; whereas, in prolapse of the kidney, we can press our fingers up under the ribs and press the mass away from them.

You will observe that we have in this case a noticeable abdominal enlargement that gives firm resistance to pressure and a dull percussion note. The first thing to determine is whether it comes from above or below. I first press my fingers deep into the abdomen above to see if it comes from under the ribs, and I find it does not. I find a free

space there. We will now feel if it comes from below. I press my hands pretty well down into the left iliac region and get a little resistance there. I come against a small solid body like an ovary, which slips from under my finger. But I cannot get any pelvic connection on this side. I feel firm resistance, however, down in the right iliac region, indicating an ovarian origin. Uterine tumors, unless pediculated, can be felt to extend deep into the pelvis just behind the pubes, rather than on one side, as in this case.

The abdominal resonance in this case is not very great, and there ought not to be much if the patient was properly prepared. However, there is some. There seems to be more gas on one side than on the other. I get resonance in the left iliac region and dullness in the right, showing that the mass belongs more to the right side than to the left. Upon auscultation we get a loose wave of fluctuation like that of dropsy, but it cannot very well be from dropsy, for the intestines would be on top and give central resonance unless held out of the way by adhesions. But in that case there would be a history of abdominal pains, due to the inflammation that caused the adhesions. Another thing we find,—namely, the fluctuating wave extends over the whole dull area, as if there were only one cyst. This kind of fluctuation, together with the flabbiness, denotes a large cyst with thin walls, possibly a parovarian cyst, or a large accumulation of fluid in the broad ligament. Most ovarian tumors are multilocular, yet some of them have a large thin-walled cyst on top. We need more definite information.

I will, therefore, make a digital examination per vaginam, and tell you what I find from below. The cervix is only two inches from the suprapubic ligament. It is rather high up,—say an inch and a half from the coccyx. You will notice that as I press the parts from below the wave passes over the abdomen, proving that the cyst goes down into the pelvis. In front of the uterus I feel nothing from below. However, as I press my other hand over the pubes, I catch the uterus bimanually behind the pubes. It is a little bit enlarged. I recognize a soft mass situated behind the uterus and unconnected with it. It is high up in and above the pelvis, and draws the uterus up with it. It must be connected with the uterus, because it draws the organ up. It is, however, not uterine, because I can move the uterus separately and get my fingers between it and the uterus. It is probably an ovarian tumor. If it were in the broad ligament, it would be against the uterus and would move with it. I cannot feel the pedicle.

You notice that I again disinfect with the bichloride solution the last thing, after handling different articles, before I begin to operate.

We shall make a median-line incision half-way between the umbilicus and pubes. If we go into the abdomen for pelvic inflammation we are apt to go low down ; but for a tumor of this size, it is better to make an incision a little higher above the pubes and enlarge it afterwards in either direction, according to what we find. Having reached the peritoneum, we draw it up with forceps and cut it between them, and enlarge the opening with scissors. I find a soft, thin-walled bluish cyst, slightly adherent to the intestines. By passing my fingers down to the uterus I find the pedicle to be the right broad ligament, and that there are some smaller projecting cysts. On account of the adhesions and smaller cysts, I will enlarge the incision to see just what I am doing. We must be careful how we handle the intestines, for every time we rub them we take off a little epithelium and prepare the way for subsequent peritonitis and adhesions. Very thin cyst walls usually contain thin fluid, so I plunge a small trocar into the tumor, and, as it collapses, draw up the sac with forceps, so as to prevent the contents from entering the peritoneal cavity. The smaller cysts come out whole. The pedicle appears in the incision, is clamped, and the tumor cut off. The pedicle is too fleshy to be safely included in the Staffordshire knot, so we will ligature it in three parts. As there is no oozing of blood within the peritoneal cavity, I close it up without drainage.

In closing the incisions of this length I use two buried sutures, about an inch and a half apart, including the peritoneum, deep fascia, and a little of the muscle to insure against hernia. Silkworm-gut becomes encysted and remains unaltered for years. I also use silkworm-gut for the external deep sutures, which include all of the tissues and are placed two-thirds of an inch apart. A superficial stitch is placed between each deep one. The deep ones are left in for one week, the superficial for two weeks, and the buried sutures forever.

THE TECHNIQUE OF LAPAROTOMY.

DIDACTIC CLINICAL LECTURE DELIVERED AT THE LONG ISLAND COLLEGE HOSPITAL.

BY ALEXANDER J. C. SKENE, M.D.,

Professor of the Medical and Surgical Diseases of Women in the Long Island College Hospital, etc.

GENTLEMEN,—The morning is the proper time to perform an ovariectomy, for the reason that an anæsthetic should always, in my opinion, be given when the stomach is entirely empty. I am sure that our neighbors in Europe have an advantage in doing their important operations early in the morning. If the patient is made to wait without food until the afternoon she is weakened by it, and if food is given in the morning before operation it is seldom digested. The final arrangement of the patient is important. It is necessary to have her clothing clean and well protected, and that can be done by using a clean rubber cloth, with an opening in it at the median line, the edges of which can be fastened to the abdomen with adhesive plaster. This rubber cloth should cover the entire patient and keep her clean, warm, and dry. I mention this rubber cloth or sheet because it is not generally employed in this country and because it is one of the little things that help. The feet should always be protected from cold. I use a warm bottle or rubber bags placed between the feet, but not in contact with them nor too close to them. I have seen half a dozen cases badly burnt during operation or afterwards by the injudicious use of these hot bottles or hot water bags.

There is much that might be said about anæsthetizing. I have long felt satisfied that many cases do badly because of bad etherization. The great advantage we gain in employing the inhaler we use here in the hospital is that the patient does not inspire the expired or foul air, whereas, with the ordinary apparatus they do. They should get pure air and pure ether, or, rather, an intermixture of pure air and ether, the proportion of ether being sufficient to keep up anæsthesia.

Everything employed in the operation should be clean,—i.e., instruments, sponges, dressings, and operators should be surgically clean or aseptic. Instruments should be washed clean and steamed in a sterilizer and kept clean and warm during the operation. The cleansing of the surgeon's hands has received much attention. In fact, some of the methods of preparing the hands are so complicated that an active practitioner hardly has time to carry out the directions. The method that I employ is simple and perfectly satisfactory. I thoroughly wash and scrub with a brush in soap and running water, boiled water being preferred. The hands are then immersed in a five-per-cent. solution of carbolic acid in glycerin and water. If I have any doubt about the soap and water, I thoroughly apply carbolic acid one part and glycerin eight parts to the hands, and after letting it remain about two minutes wash it off with distilled water or water that has been filtered and boiled. Hands treated in this way are clean and can be used with absolute safety. The sponges should be made sterile and kept moist and warm.

In former times surgeons used to operate in a very high temperature, almost up to 100° F., which was very unpleasant both for the operator and the patient, but it was deemed necessary to avoid exposing the abdominal cavity to cold air. Now the patient is protected from cold and shock with hot sponges, just as hot as can be comfortably handled. Also see that your hands are warm. It is necessary, during a large operation, to put big sponges into the abdomen. Besides protecting the patient from shock, these sponges check hemorrhage and keep the intestines from coming down. We keep the sponges in a pail, which is put into another pail; the inner one is thoroughly sterilized and filled with sponges, and the outer one is filled with hot water, and a lid is put over both. The sponges are removed when used. As a dressing for the abdominal wound I use gauze saturated in a solution of carbolic acid and glycerin (one to eight); the glycerin takes away the caustic properties of the carbolic acid. The gauze is thus thoroughly disinfected. The cloth is wrung out immediately before it is applied. It is spongy and porous, and any oozing of serum will be mopped up and disinfected. Decomposition cannot take place, and the surface is kept moist and not at all irritated. Then, to protect it from outside influences, I apply sterilized cotton and a bandage.

Now let me say a few words about the operation itself. Ovariectomy may be an exceedingly simple operation, as simple as any of the major operations in surgery, or it may become, and often is, the most

difficult in the whole range of surgery. In nearly all other operations that you are called upon to perform you can qualify yourselves beforehand. I cannot at this moment recall a single operation, except ovariectomy, that you cannot practise on the cadaver or manikin; but one cannot make a manikin or get a cadaver that presents all the complications that may occur in ovariectomy. It is necessary to have in mind very clearly all the conditions that may occur, and to have seen a number of such operations performed. Otherwise it will prove exceedingly difficult, especially in those complicated cases. I can give you no help by describing the different steps of the operation, nor is it necessary, as you can get that information from any of your books on the subject. I will simply give you a few hints which may assist you. Every operator, when he describes the method of operating, describes his own, and it may not be the best. The trouble with all these descriptions is that they lack the details,—the little things which one needs to know.

First, as to the method of opening the abdomen. Always choose the median line. Make the first incision quickly, going through the skin and the subcutaneous tissue, down to the muscular tissue. Then find the median line between the two recti muscles. That is not always easy, but by making an incision through the fascia, and passing a blunt scissors first one way and then the other under the sheath of the muscle, the median line is found. When the peritoneum is reached raise it up, if it is not adherent, with the forceps; make a little opening in it with a blunt-pointed scissors, and then with the scissors divide it. With reference to tapping or drawing off the contents of the sac, there is nothing in that which you will not get clearly from your books, except that the trocar canula used differs with each operator. For the average operator the best trocar is Keith's, which I here show you. The great object is not to allow the fluid to get into the wound or back into the abdominal cavity. While the fluid is running off seize the sac with compression forceps and draw it up into the wound first and then out. When there are multiple cysts we are advised to tap one from the other, which is exceedingly difficult to do. I have adopted this plan: After tapping one sac I take out the trocar and close the wound with a locked forceps; then I tap the next one that comes within reach. By repeated tapplings from the outside you are sure that you will do no harm. Sometimes you will plunge in your trocar and tap one cyst, and then you can find no other cysts large enough to tap; it is just a mass of little cysts, or the fluid is so thick that it will not flow. Such a condition of things is exceedingly embarrassing. It is not so bad if there is one cyst that you can empty, because then you can draw

it out and pass your hand into the sac, and break down and scrape or scoop out the whole contents, holding the rubber cloth up to carry them off. Some operators roll their patients over on the side while emptying the cyst. I have the patient strapped to the table-top, which is movable, and roll patient and table-top to one side. When you reach the pedicle ligate it and close your wound. Stitching the wound will sometimes bother you, unless you remember, as soon as your sac is drawn out, to insert a sponge in the abdominal cavity to keep the intestines out of the way. It keeps the abdominal contents out of the way and absorbs any blood that may come from your needle puncture.

There are two ways of treating the pedicle. One is by means of the cautery clamp and the other by ligature, just as you would ligate an artery. Either catgut or silk ligatures are used. I am always afraid of catgut, because it is difficult to get it perfectly clean and is very apt to soften and let go. I prefer the silk ligatures. The cautery clamp is preferable to ligatures, because it controls all bleeding and there is no foreign body left in the wound, but it requires a great deal of skill and practice to use it properly. It takes more time, and when you take off your clamp you are always afraid that something will happen.

The most important complication that we encounter in ovariectomy is adhesions. I need only say a word or two on that subject. For a long time, and even to-day, many operators, when they have exposed the sac, and before tapping it, put in a male sound and break up or separate any adhesions that may exist in front. That is the greatest mistake that one could possibly make. One should never separate or break up an adhesion without seeing it or feeling it. When you find that your sac is adherent to the abdominal wall, always with your fingers separate enough around the wound so that you can draw the emptied sac out of the wound, and as this is being done, you should find the adhesions, ligate them first and then separate them. Ligate them in mass or in sections, according to their extent. The first thing usually encountered is adhesion of the omentum. Ligate it in sections, by using one ligature to avoid splitting or tearing the omentum. Adhesions to lateral portions of the abdominal wall can be broken up with the hand, or sponged off. Intestinal adhesions should be treated in the same way as omental adhesions. When adhesions deep in the pelvis are found they must be separated with the fingers. Afterwards see if there are bleeding points left, and, if so, ligate them. There will be a little serous oozing sometimes which you cannot stop, but actively bleeding vessels should be ligated. You should operate

quickly, so as not to expose the abdominal cavity too long. That is why the wisest of all operators said that no man should perform an ovariectomy until he has had a good deal of experience with living tissues and bleeding vessels. As to ligaturing adhesions, unless the adhesions are exceedingly thick and strong you will find that catgut will answer the purpose. I use silk, prepared as I have directed, to ligate the pedicle and to suture the abdominal wound.

When the tumor has been removed and the abdominal cavity is left clean and dry, the wound may be completely closed, with a certain confidence that all will be well. When there is reason to suspect the presence of septic material,—in case, for example, where a portion of the fluid has found its way into the peritoneal cavity; or in case there are many adhesions, and therefore bleeding vessels which require to be ligated, so that a number of ligatures are left in the abdominal cavity, or surfaces from which adhesions have been separated, that are not bleeding, but yet are raw,—I say when any of these conditions are present drainage is necessary. Much has been said for and against drainage; yet I am sure that whenever the conditions which I have mentioned are present, drainage is necessary and highly essential. What are the objections to the introduction of a drainage-tube? It is a foreign body in the abdomen, and acts, to a certain extent, as an irritant. It keeps a part of the abdominal wound open and the wound does not always unite immediately after the tube is removed, hence the patient is more liable to ventral hernia. Again, a drainage-tube causes an accumulation of inflammatory products which must be disposed of afterwards, prolonging convalescence and increasing the number of after-adhesions, and the patient is more likely to have intestinal obstruction.

All these objections are overcome to a great extent by not prolonging drainage to over twenty-four or forty-eight hours. With reference to its interfering with the possibility of perfect union, that is largely avoided by using a small drainage-tube. A small one answers the purpose just as well as a large one,—in fact, better. I usually prefer a perfectly straight glass tube with a flange at the upper end and with perforations in two-thirds of its length.

I must say a word about the management of the drainage-tube. The rule with some operators is to put in a big tube, cork it up, and empty it from time to time. The proper way is to pass the tube through a piece of rubber tissue, then leave it open and put a perfectly clean sponge over it, and tie your rubber cloth over that. The smaller your tube within certain limits the more perfect will be your drainage. The

• pressure of the abdominal walls and the capillary attraction keep up a continual welling. Just as soon as all oozing ceases, and all that can be brought out by careful pumping is a little, almost clear, serum, take out your tube. If the serum is still colored and there are flakes of lymph in it, let the tube remain. By that kind of drainage I am sure many lives are saved.

Then again, in cases where there is secondary hemorrhage the flow from the tube announces it, and the surgeon at once sees the indications to open the abdominal cavity again and control the bleeding vessels.

PESSARIES.

CLINICAL LECTURE DELIVERED AT THE BUFFALO GENERAL HOSPITAL.

BY MATTHEW D. MANN, A.M., M.D.,

Professor of Obstetrics and Gynæcology in the Medical Department of the University of Buffalo.

GENTLEMEN,—The patient that I show you to-day has come here for the purpose of having her pessary looked after. She was under my care in the hospital some three months ago. Previous to that time she had had scarlet fever, and after she became convalescent it was found that she was not getting along as well as she ought. She was unable to walk or stand, and she had a great deal of backache, from which she had suffered for some time before the attack of scarlet fever. On my first examination I found the uterus decidedly retroverted and quite small. It lay high in the pelvis with no indication of prolapse. One of the attending physicians under whose care she had been said to me, "You do not expect to do anything for that case by treatment with a pessary, do you?" Now, I have brought the patient into the clinic to-day so that she may tell you what the result of treatment by means of a pessary has been. She says that she is working hard as a house-maid and has had no trouble at all, the backache, pain in the side, and discomfort having disappeared. Her monthly periods have been regular; they are quite painful, but she is not obliged to give up work at that time. She now complains of shooting pains in the chest, undoubtedly the symptoms of intercostal neuralgia, which is a very common accompaniment of uterine disease, and which is due, I believe, to an indirect connection between the pelvic and intercostal nerves.

I am glad to have the opportunity of showing you this case, because it illustrates very well what can be done by treatment with pessaries. There is a great deal of incredulity in the medical profession at large with regard to the good results which can be obtained from the use of pessaries. Without pessaries, I should not know what to do for a considerable number of the cases that come to my office, and I should almost have to give up gynæcology, although I might continue to do

laparotomies. I do not believe in pessaries for anterior displacements, but I do believe that they will effect a cure in a considerable proportion of cases of posterior malpositions, and will relieve the distressing symptoms in a still greater number. Only a small proportion require surgical treatment. This woman felt better within a few days after the insertion of the pessary, but I kept her in the hospital for probably a month, being obliged to change the pessary two or three times before I got one that was exactly of the right size and shape to hold the uterus in place; the pessary used was an Albert Smith retroversion pessary. When I secured this result the patient rapidly improved, her pains left, she was able to help about the ward, and in a little while she was discharged. This is only one of a great many cases that I could show you, to illustrate how women have been made absolutely well simply by wearing a pessary.

Why is it, you will ask, that there is such a strong prejudice in the medical profession against the use of pessaries? Emmet says that the man who gets bad results from pessaries does not know how to use them, the fault being in the man and not in the pessary. Thomas makes a similar statement. Skeene has a chapter on the abuse of pessaries, but he does not give them up on that account. Goodell agrees substantially with the others. Pessaries do harm because they are not properly fitted, and because they are not properly cared for afterwards. In the first place, the case must be a suitable one for the use of a pessary. There is no use putting a pessary into the vagina if the uterus is bound down by adhesions; the uterus must be replaced before the pessary is used. The pessary must not be expected to act as a repositior, and even if there are no adhesions holding the uterus down, the uterus must be in correct position before the pessary is inserted. Again, the pessary must be properly fitted. I have had to refit a great many of them, and have found that the very common mistake is made of relying on a pessary which is too short. To appreciate this fault, you must understand the mechanism of their action. There have been volumes, almost, written on this subject alone. Let me illustrate by this sound a uterus in retroversion, my fingers representing the place at which the ligaments are attached. As you see, I can tilt the sound forward either by pressing forward on its upper portion or backward on its lower portion; in case of the uterus I cannot do this, for I cannot get my fingers behind the fundus to tilt it up without performing a laparotomy. But we can use the cervix as a handle to replace the organ, provided the uterus is rigid enough. The pessary goes in behind the uterus; it does not, however, as might appear, press upward

on the fundus, for it is anatomically impossible for the pessary to push up so high in the posterior fornix of the vagina. I have opened the abdomen when the uterus was held in place by a pessary, and have been able to demonstrate that the pessary was not in contact with the uterus at all; I could even put my finger between the uterus and the pessary. The pessary acts simply by pulling up on the posterior vaginal wall, and thus indirectly drags the cervix upward and backward so as to act in the same way as if we had pushed on the front of the cervix. Now, you will understand from this explanation why it is that when a pessary is too short it will utterly fail to do good, for it does not pull the cervix backward and upward far enough. Moreover, if the pessary is not long enough, its upper bar does not reach high enough to push the uterus past its centre of gravity and the version will be converted into a flexion; the uterus, so to speak, being doubled over the bar of the pessary. But if the uterus is pulled over far enough, this cannot occur. The pessary must be adapted to the vagina, and this organ differs as much in different individuals as any other organ.

Do not rely on the pessary to replace the uterus; first reposit the uterus and then insert the pessary, having it long enough to keep the uterus in its normal position. One of the roughest-looking specimens of the medical profession that I ever saw was a country practitioner who knew more about the use of pessaries than almost any other physician I have ever met. He failed in one case simply because he did not know he could get a pessary large enough. He understood perfectly what the trouble was, and he was delighted when I opened a drawer in which my pessaries are kept and handed him one not quite as large as a horse-collar. I gave him a few like it, and he was enabled to meet the indications in his case. A man must have a little natural mechanical skill and taste in order to use pessaries properly; and, in fact, a man without mechanical ingenuity ought not to undertake gynaecological practice.

In regard to this present case, I must state that in spite of the favorable account which the patient gives of herself, the uterus is found on examination not to be thrown quite far enough forward. The pessary gives marked relief, because the uterus is thrown forward sufficiently to prevent its becoming engorged with blood; but in order to accomplish a cure, I must insert a still longer pessary so as to throw the uterus still further forward into anteversion, the natural rebound of the tissues being sufficient to bring about the happy medium after the pessary has been removed.

THE TREATMENT OF UTERINE CANCER.

CLINICAL LECTURE DELIVERED AT THE NEW YORK CANCER HOSPITAL.

BY HENRY C. COE, M.D., M R.C.S.,

Professor of Gynecology at the New York Polyclinic; Gynecologist to the Cancer Hospital; Obstetric Surgeon to the Maternity Hospital; Assistant Surgeon to the Woman's Hospital in the State of New York.

GENTLEMEN,—Our first patient is fifty-nine years of age, and passed the menopause ten years since. About three years ago she had a slight irregular "show" of blood, and at intervals of several months she has had slight hemorrhages. You remember that I have spoken to you before about this as being an ominous symptom. There has never been any foul discharge in this case, nor any pain. Moreover, the general health has been but little affected. A fragment of tissue is said to have been removed from the cervix and examined some time ago, and was reported to be non-malignant. She has on several occasions been etherized and the uterus curetted and cauterized. The fact that the trouble had extended over a period of three years led me to suspect the existence of malignant disease, although her health is so good. According to my usual custom I examined her thoroughly under ether last week, in order to determine if the uterus was movable and if there were any evidences of metastatic deposits, and at the same time settled the diagnosis of cancer with the aid of the curette and microscope. Having determined the fact that there were no local or general contra-indications to a radical operation, it was clear that there was nothing to be done but to remove the entire uterus, although from the length of time during which the disease has probably existed, I do not feel very hopeful with regard to long freedom from recurrence.

There are several conditions which indicate at the outset that the operation will be a difficult one. Although the uterus is small, and no secondary infiltrations can be detected in the broad ligaments, its mobility is limited, leading to the inference that there are fundal adhesions. The vagina is so narrow that it will be difficult to use ligatures,

so that I shall apply clamps temporarily. Having disinfected the uterine cavity and vagina thoroughly, I pack the former with iodoform gauze, then seize the cervix with a volsella and apply strong traction downward, while I make a circular incision around it. The rest of the dissection is done mainly with the forefinger, the bladder being separated from the uterus as high as the vesico-uterine fold of peritoneum. The peritoneum forming Douglas's pouch does not extend as low down as usual and is much thickened, so that it is difficult to identify it. As soon as I have incised it I introduce a gauze-pad (to which a string is attached), both to prevent prolapse of a loop of intestine and to absorb any blood which may escape into the peritoneal cavity. Now, making strong traction downward, I clamp the base of each broad ligament in turn, including the uterine artery, and divide the included tissue close to the uterus. There is more inflammatory induration on one side than I had supposed, though it does not present a suspicious appearance. The uterus is still held by firm adhesions above. I now draw down and open the anterior fold of peritoneum, when I am able to hook my finger over the top of the left broad ligament and to apply another clamp, which includes also the tube and ovarian artery. After dividing the rest of the ligament on this side I can hook down the fundus and, after separating a few adhesions, draw out the entire uterus, which is now held by the right tube and the upper half of the corresponding broad ligament. These are clamped, divided, and the organ is removed. You observe that I have removed only the left ovary and tube; the adnexa on the right side are not only firmly adherent, but are so high up that I can barely touch them, so it seems wiser not to disturb them.

This has not been a favorable case, and had I known that the operation was going to be so difficult, I doubt if I would have attempted it. Those of you who were present three weeks ago when I performed vaginal hysterectomy for circumscribed malignant adenoma of the corpus uteri, will remember that the operation was very simple and easy, quite the reverse of this one. In the former case the vagina was large and the uterus perfectly movable; here the organ was firmly adherent and there is limited working-space. As there is more room now, I shall ligate the stumps and remove the clamps. I no longer leave these on for forty-eight hours, as I did formerly. This is a favorite method with French surgeons, but does not impress me as being entirely safe or surgical, while the patient is certainly much more uncomfortable than she is after ligatures have been used. I have known of cases of fatal secondary hemorrhage when the forceps were removed on the second

day. Then, too, there is extensive sloughing of the tissues and more risk of sepsis. I lost two patients from intestinal obstruction (doubtless of septic origin), due to retraction of the stumps and adhesion of adjacent coils of intestine after removal of the clamps; the stumps were also not properly covered with gauze.

I now bring the ends of the four ligatures outside of the vulva, and having satisfied myself that all oozing has stopped, introduce a gauze tampon in the following way: Exposing the wound clearly with the aid of lateral retractors, I pass a narrow pad of iodoform gauze well up into the peritoneal cavity, so as to cover the stumps on the left side; place a second piece over the opposite stumps, and, holding the two apart, fill in the space between with several feet of gauze bandage. A pad over the external genitals and a firm T-bandage completes the dressing. The central strip of gauze will be gradually drawn out after three or four days, but the side-pieces may not be removed before the seventh or eighth day. There is no hurry about detaching the ligatures; the last one may not come away for two or three weeks. Although the operation has been prolonged to nearly an hour, there has been but little loss of blood, and the patient ought to make a good recovery; though, as I said before, I fear that the ultimate result will not be satisfactory.

Our second patient is thirty-six years of age, has been married seventeen years, and has had good general health. Her menstrual history is normal. The first symptoms of her present trouble were noticed in December, 1890, when she began to have a muco-purulent discharge at the end of micturition, accompanied by sharp pain at the neck of the bladder and tenesmus. This increased in severity, and she then began to pass blood with the urine. She was treated for several months at a dispensary for cystitis, her bladder being frequently washed out, but without benefit. She was sent in as a case of possible malignant disease. The house-surgeon, who examined her for admission, detected a calculus, which I propose to remove. The bladder has been thoroughly washed out, and repeated examinations of her urine have failed to show any renal complications. I shall perform cystotomy in order to satisfy myself both by touch and by inspection regarding the condition of the vesical mucous membrane. If there is no cystitis, the wound can be closed at once; but if the mucous membrane is much diseased, drainage will be established. Calculus is not very common in the female, as compared with male subjects, and when the stone is small it is occasionally expelled through the urethra. Some surgeons recommend dilating the urethra and removing the calculus through this chan-

nel; but this operation is very apt to be followed by incontinence of urine, and it is better to perform cystotomy. I propose in this patient to establish a vesico-vaginal fistula in the bladder by stitching together the mucous membrane of the bladder and that of the vagina. The patient is placed on the side, a sound is introduced into the bladder, and the presence of the stone is determined beyond question, as you hear the distinct click when it comes in contact with the instrument.

Now, turning the point of the sound backward so that its tip is felt just below the cervix, I cut down upon it and soon enter the bladder. The opening is enlarged with angular scissors, so that I can introduce my finger and thoroughly explore the interior of the viscus. The stone is about the size of a marble and is easily grasped with the dressing forceps and removed. It is, as you see, of phosphatic character, and is quite friable. Now, palpating the interior of the bladder again, I find that the mucosa is not only much thickened, but is coated with gritty material which may readily form the nuclei of other calculi. After irrigating thoroughly with warm boric acid solution, I pick up the vesical mucous membrane and stitch it in the edge of the vaginal wound with a continuous suture of fine silk. I shall drain the bladder for a month or two, until I feel sure that the cystitis has been cured and that the fistula may be closed without a return of the former trouble.

The third case is one of malignant disease of the body of the uterus, which I fear is so advanced that a radical operation will be impossible. This can be determined by an examination under ether, when the curette can also be used thoroughly, if nothing more can be done. The patient is forty-five years of age, and had enjoyed good health up to the commencement of the present trouble. She has not menstruated for three years. One year ago she had a hemorrhage lasting a week. Since then there has been a purulent vaginal discharge with some odor, associated with severe pain in the back, thighs, groins, and with marked emaciation. She has a very small vagina and a large uterus, which is absolutely fixed. There is no doubt, from the history and from her general condition, that malignant disease is present. Having placed her in Sims's position, I dilate the cervix with a steel instrument and irrigate the uterine cavity thoroughly with Thiersch's solution, before introducing the sharp curette. You see that I use a simple glass tube for irrigation, and it answers the purpose better than the more elaborate double canulæ. I have never seen any evil consequences from the use of such a tube, for there is no danger of injecting fluid into the Fallopian tubes as long as there is a free outlet, and you should never

wash out a uterus unless you have dilated the cervix sufficiently to secure a return flow. In this case we find that the disease is confined to the fundus. I have removed a large quantity of soft, brain-like material. You notice that the rather free hemorrhage is promptly checked by the hot intra-uterine douche, after using this I swab out the cavity with pure carbolic acid and pack it with iodoform gauze. The uterus is quite large and is absolutely fixed, while the general induration of the peri-uterine tissues proves that the disease has extended beyond its original site. Abdominal hysterectomy is, of course, entirely out of the question. The prognosis is absolutely bad, and there is nothing to do for the patient but to make her as comfortable as possible during the short time that remains to her.

VILLOUS ENDOMETRITIS; DISPLACED KIDNEY; PREGNANCY DURING ENDOMETRITIS.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POLYCLINIC.

BY DR. PAUL F. MUNDE,

Professor of Gynæcology at the New York Polyclinic and at Dartmouth College.

VILLOUS ENDOMETRITIS.

GENTLEMEN,—This patient is twenty-seven years old ; she has been married nine years, and has had three children, the last one about five years ago. She comes here to-day complaining of profuse and painful menstruation. The flow comes on every three weeks and lasts for six days. In addition to this she complains of pain in the right ovarian region, and there is a profuse white vaginal discharge. She has a poor appetite and digestion, and suffers from constipation, and the frontal headache which usually accompanies it. The pain in the lower part of the abdomen has lasted for about one year.

It is not probable that her pelvic pain depends upon the last confinement, on account of the long interval which has elapsed. On examination I find the uterus in the normal position, except that it is a little low in the pelvis ; it is slightly ante-curved, the cervix is broad, the external os gapes so as to admit the index finger, and the uterus is moderately enlarged, but not enough in itself to be of much significance. Both ovaries can be easily felt ; they are nodular and evidently contain a number of distended Graafian follicles. The tubes are apparently normal, hence there is nothing in the uterus or the adnexa which will explain her profuse and painful menstruation. We must, therefore, place the patient on her side, and, by means of the blunt wire curette, ascertain whether there are any small vegetations or polypoid growths in the uterine cavity, which might account for the profuse menstruation. The discharge from the vagina is of a thick viscid character, and this would indicate the presence of cervical catarrh. As it is neither yellow nor profuse, I do not attach much importance to it. We frequently meet with such cases in which the physical examination, and even one made with the speculum, does not reveal any reason

for the profuse menstrual flow ; and under these circumstances the only thing left for us to do is to look for these vegetations. These are usually the result of chronic endometritis ; that is, a hyperplasia of the normal glands of the mucous membrane, in consequence of chronic congestion. I am very glad to be able to show you a woman in whom the ordinary examination shows nothing abnormal, because this will enable you more readily to determine pathological conditions when you meet with them. There is some difference among authorities as to the exact normal position of the uterus. This is because the organ is movable and its position may vary within certain narrow, but perfectly normal, limits. The slight laceration of the cervix in this patient is of no consequence, excepting that it might leave the cervical canal so widely open as to give rise to a hyperæmia of the cervical mucous membrane, resulting in the production of a profuse discharge. Now, before passing the curette, you should ascertain the direction of the uterine canal, and this may be done by the introduction of either the uterine sound or the uterine probe. I do not think the size of the sound or probe is of much importance ; it is the gentleness of your manipulation that is the essential point. Having ascertained the direction of the canal, the blunt wire curette is made to correspond with this curve, and is then passed up into the uterus and gently moved down two or three times. Examining carefully on a piece of cotton whatever may be discharged as a result of this manipulation, you can ascertain whether there be any of these small vegetations present. There is always a certain small risk attendant upon even this simple manipulation, but it is justifiable on account of the important diagnostic aid which it gives. We find here a few of these small growths, and as one passes the curette over the endometrium it gives a pulpy sensation, which indicates that there is a hyperæmic condition of the endometrium. Even if the vegetations had not been removed, this peculiar sensation imparted to the hand while manipulating the curette is sufficient to make a diagnosis.

Of course, the treatment of such a case is to thoroughly curette the uterus ; but I do not care now to take the risk of doing this in dispensary practice. Formerly, I advocated its use even in these cases, but I have seen serious consequences following this practice, and now would not advise it. You may object that we have scraped away with the curette the sound mucous membrane. My answer is, that unless you do this curetting just before a menstrual period, you cannot remove such particles from the perfectly normal mucous membrane. Dr. Goodell says that the best time for doing this curetting is *during* the menstrual period. This is, perhaps, going rather too far ; but I

certainly would not allow an impending menstruation to prevent me from doing a laparotomy, for I have often done this and have never seen any bad results. It is usually not necessary to employ an anæsthetic, unless the patient be excessively nervous and hyperæsthetic. The patient should always be put to bed immediately afterwards, and my present practice is always to put an ice-bag on the abdomen for twenty-four hours, simply as a matter of precaution, for, if there be any tendency to inflammatory reaction, such treatment must certainly tend to diminish this. After curetting, it is my routine practice to swab out the uterus with compound tincture of iodine, taking care to bring the fluid in contact with the whole cavity; then a thin strip of iodoform gauze is passed through the internal os, and the vagina is loosely packed with the same material. A secondary hemorrhage is hardly to be feared if the iodine application has been thoroughly made. After the curetting do not think your work is done, but keep on making applications of tincture of iodine to the endometrium; at first twice a week, and afterwards less frequently, until two or three menstrual periods have been perfectly normal. The reason for this is that these vegetations are very apt to return.

DISPLACED KIDNEY.

This patient is twenty-five years of age, is a dress-maker by occupation, and is unmarried. For four years, but chiefly during the past month, she has suffered severe pain in the left iliac region, and also higher up towards the umbilicus. She also complains of pain in the back part of the left hip. She came to me a few weeks ago, and I found her pelvic organs apparently normal. Having in mind a possible dislocation of the kidney, I asked her to cough and strain; and upon her doing this, I thought I felt an organ corresponding in size and situation to the left kidney in the painful region. I advised her to go to an instrument-maker and have a pad applied. She was advised by friends to seek medical advice elsewhere. One eminent physician discovered a dislocation of the right kidney, and a very well-known surgeon found nothing at all. In view of this conflicting testimony, I thought it best to make another examination.

Placing the patient in the sitting position, I find that palpation of the left side of the abdomen causes pain, and, one hand being placed in front and the other behind, I can distinctly feel a movable mass on quickly tapping the abdomen. Displacement of the kidney is much more common in women who have borne children. To settle the diagnosis, it would be proper to examine this patient under ether.

PREGNANCY DURING ENDOMETRITIS.

This next patient is twenty-six years of age ; she has been married three years, but has never been pregnant. She flowed every four weeks for four days, until four months ago, since which time she has been "spotting" irregularly. During the last three weeks she has been flowing constantly. She complains of pain in the abdomen, and there is a profuse white discharge.

On casual inspection the hymen seems to be perfect, but closer examination shows that it is torn on the right side. This is the kind of a hymen which indicates coition, but not parturition. This point might in some cases assume great medico-legal importance. This woman has had no children, and, therefore, has no lacerated cervix. This "spotting" in all probability is due to a chronic endometritis. You will probably find, on digital examination, a slight bloody discharge and the lips of the external os soft and pulpy, and, perhaps, the external os slightly gaping. You notice that, although I have used no instrument and my finger nails are short,—as they should be with every gynaecologist,—on withdrawing the examining finger it is covered with blood. This leads me to think that we shall find an eroded vascular cervix. The uterus is enlarged to the size of my fist, more particularly towards the right side, and its softness is very suspicious of pregnancy. Her history is not by any means incompatible with pregnancy. The chances are that the woman has become pregnant some time since the "spotting" began, because the uterus does not correspond to a pregnant uterus of more than three months. There was probably a cervical catarrh before she became pregnant, and this condition has increased during the pregnancy. This woman may, of course, miscarry at any time, although it is by no means necessary that these symptoms should be followed by such a result. Let me remark, in passing, that when making a careful vaginal examination at a patient's house, I invariably insist upon her being placed across the bed with the knees drawn up, so that the parts are thoroughly relaxed.

As the enlargement in this case is apparently not due to a fibroid or a myoma, it is more than probable that it is due to pregnancy.

VAGINAL HYSTERECTOMY; CURETTAGE OF THE UTERUS.

CLINICAL LECTURE DELIVERED AT THE HARPER HOSPITAL.

BY H. W. LONGYEAR, M.D.,

Professor of Clinical Gynæcology in the Detroit College of Medicine.

GENTLEMEN,—The case on which I am about to operate is one of carcinoma of the cervix uteri. The patient's history is as follows: She is thirty-seven years of age, married fourteen years, and is the mother of three children, the last one having been born dead six months ago. Her family history is good. Menstruation has always been normal. For nearly a year she has complained of a feeling of bearing-down and weight in the pelvis, besides a steadily increasing condition of weakness and lassitude. She has noticed a thin watery discharge, with very little or no odor to it, for about six months, and that intercourse causes hemorrhage. It was principally because of this latter symptom that something wrong was suspected by the patient. My friend, Dr. R. A. Newman, being consulted, immediately recognized the nature of the disease and sent her to me for operation.

When a patient comes to you with a history similar to this, you should immediately make a careful examination of the uterus and its surroundings, as an early diagnosis in this class of cases is of the utmost importance. *In cancer of the uterus, life can be saved only by early operation.* I want to impress this fact on your minds, as the lives of many of these unfortunates are being constantly sacrificed by the treatment of procrastination. In cancer of the neck of the uterus there should be rarely any difficulty in making the diagnosis; or, at least, in *suspecting* the nature of the disease sufficiently to give the patient the benefit of the doubt by early counsel, microscopical examinations, etc. There is no excuse for the physician who, either through ignorance or cupidity, goes on for months treating by local applications such a case as this without acquainting the patient with the nature of her disease. There are conditions that might reasonably excuse a phy-

sician from making a diagnosis or suspecting the presence of cancer of the uterine body, but I know of none that should permit him to treat for months a constantly increasing malignant growth situated in the neck of the uterus, and yet be ignorant of its nature. The feel on digital examination is usually quite characteristic: the edges of the diseased part are very hard and somewhat raised, while inside of this circle of infiltrated tissue the surface will have become softened, so that in many cases you can easily detach particles with your finger. This ulcerated part bleeds freely on touch, and is the portion from which the discharge of blood and pus comes. This patient has been treated for nearly a year by a physician in this city who evidently did not recognize the nature of the disease, as the patient was not aware of it until informed by Dr. Newman one week ago. The uterus is considerably enlarged but freely movable, indicating absence of malignant deposit in the broad ligaments or appendages. There is also no indication of extension of the disease to the bladder or rectum. I think that the entire removal of the organ will give this woman a very fair chance of escaping death by this terrible malady. However, notwithstanding the fact that we find no evidence of extension of the disease beyond the uterus, some of the lymphatic glands outside of this organ may have already become affected by the long continuation of the disease; and if this is the case, the condition will return and our operation will have been in vain. This is a point that you must not lose sight of in making a prognosis and in stating the chances of recovery to the friends of the patient.

I shall remove the uterus through the vagina by the operation known as vaginal hysterectomy. There are two methods commonly used in these cases, each having for its especial object—besides the removal of the diseased organ—the prevention of hemorrhage, and differing only in the means used. The first is by the use of the clamp, and is the method most generally used in this country. The second is by the use of the ligature. I illustrated the latter method by an operation before the class about two months ago, the patient afterwards making a perfect recovery. The technique of the two methods is as follows: After thoroughly cleansing the vagina, the surface of the diseased parts, and the cervical canal with a 1 to 200 corrosive sublimate solution, the cervix is grasped with strong volsella forceps in such a manner as to hold the os externum closed, and drawn down as far as possible. An opening is then made into the posterior cul-de-sac and enlarged from side to side to each broad ligament. Next, a corresponding incision is made, anterior to the cervix, through the vaginal wall and

cellular tissue to the uterine tissue, which is also enlarged from side to side so as to meet and become continuous with the posterior incision. The bladder is then dissected away from the uterus with the finger and scissors, and an opening made through the peritoneum. When the bladder is entirely freed the only attachments of the uterus will be those of the broad ligaments. These are either clamped or ligated and the uterus cut away. Each ligament may be clamped *en masse* by one instrument, or the clamps may be put on in detail, two or three on a side, cutting after the application of each instrument. When the ligature is used the first two are so placed—one on each side—as to control the uterine arteries, when the uterus is severed from the lower part of its attachments. The fundus is then grasped with a tenaculum through the posterior opening, and the organ completely retroverted. This brings the upper part of the broad ligament, with the Fallopian tubes and ovaries, within easy reach, when they are also ligated and the remaining attachments cut away. The operation is completed by packing the vagina with iodoform gauze, which is left for about thirty-six hours. If clamps are used they are removed with the gauze. In this case I shall use the method of ligation, unless I find the stumps too thick to be treated in this way.

The patient now being fully under the anæsthetic and the Clover's crutch applied,—which you see is of great service in holding the thighs in position,—I first draw down the uterus with the tenaculum and thoroughly disinfect all the parts with this sublimate solution; then with this strong volsella I grasp the cervix back of the diseased portion and draw it well down and forward. Now, as Dr. McLarty holds this firmly in position, with these small tooth-forceps I catch the vaginal wall near the back of the cervix, and drawing it forward form a fold which I now cut through with the scissors, being careful to keep outside of all diseased tissue. This opens the cul-de-sac, and, placing my finger therein to act as a guide, I cut freely through vaginal wall and peritoneum until the broad ligaments are reached on each side. I will now make the anterior incision, and as the cervix is pushed downward I pull up a fold of vaginal wall and cut through it as before, taking care here, however, to hug the uterine tissue closely. I now enlarge this incision on each side, going through the vaginal wall only, until it is continuous with the posterior incision. The cervix you will now see is completely encircled by the incision. Only the posterior part of the incision now enters the peritoneal cavity, and to attain the same result in front the bladder must be separated from the uterus and the peritoneum entered where it is reflected over the bladder. This I

shall now do, using my finger where possible and the scissors occasionally, always cutting towards the uterus. It is very easy to cut or tear the bladder at this stage of the operation, so that this work has to be performed with great care. Now I have stripped the bladder entirely away from the uterus and reached the peritoneum, but I am not able to enter the cavity, as my finger slides over the membrane without being able to puncture it, so the cavity must be entered in another way. Removing my finger, I insert it into the posterior opening, and hooking it over the right broad ligament from behind,—which is not an easy thing to do here, as the structure is very thick and unyielding,—I press the end of my finger down into the anterior wound close to the right side of the uterus. This puts the peritoneum on the stretch at this point, so that I am sure it can be quite easily and safely punctured with this pair of blunt forceps, which I now pass, closed, into the anterior opening, and pressing it against my finger it easily enters the peritoneal cavity. This opening I now enlarge with my fingers across to the opposite side. The uterus is now free of all attachments but the broad ligaments, so that I may now proceed to apply the first ligature, using for the purpose this strong kangaroo tendon material, and placing it by means of this long hook-shaped, blunt needle, threaded with silk. First passing the left forefinger on the upper and the thumb on the under side of the left broad ligament, I find a thin portion well back from the uterus, then along my thumb I pass the needle and force it through to the finger, which finds and pulls through the silk loop, through which I now pass the ligature and pull it into place. As I tie the knot I push it as far away from the uterus as possible, so as to leave a good button to prevent slipping of the ligature after the uterus is cut away. The tissue composing this ligament is unusually thick, so that I am in some doubt as to the utility of the ligature. However, we will try it, and if hemorrhage occurs the clamp can then be used. It now being firmly tied, I will cut the uterus away on this side as far up as my ligature goes, using these strong blunt-pointed scissors and keeping close to the uterus. I see no hemorrhage from the severed arteries, which are plainly visible, so the opposite side will now be tied off and cut away in like manner. Now that this is cut away, you notice that the uterus is much more movable, so that I shall probably experience no difficulty in retroverting the organ, which I will now proceed to do. The volsella will first be removed and the cervix again disinfected. I now place this retractor deep in the cul-de-sac, giving me a view of the fundus uteri, which I will now grasp with the double tenaculum and pull down. It comes back with some difficulty, but by

pushing the cervix back under the pubis I am enabled to completely retrovert the organ. You now see the fundus protruding from the vulva, and as I drag down the ovaries and Fallopian tubes they are seen to be comparatively healthy. The remaining portions of the broad ligaments—that is, the parts that you now see containing the ovaries and Fallopian tubes and supplied by the ovarian artery—will now be tied off with the ligatures placed outside of the appendages, as I shall remove these organs with the uterus. I find the parts to be ligated are so thick and vascular that it will be necessary to use two ligatures on each side instead of one, as I intended. Now that I have placed the first one, tying it as far from the ovary as possible, I will cut this part away. The last ligature on this side, which I am now placing, embraces all the tissue between the lower end of the incision that I just made and the upper end of the incision below. I now cut away the last attachment of the uterus on this side, and as I complete it there seems to be no hemorrhage. I must warn you that while parts are kept in a state of tension, as is the case here, bleeding is not liable to occur anyway, so that we may yet have hemorrhage after the parts have been allowed to retract. The opposite side will now be tied and cut off in like manner, using two ligatures, as it is also very thick. As I sever the uterus from its last attachment I cut close to it, leaving as large a button of tissue in front of the ligature as possible. It is very easy, while the parts are on the stretch, to make the mistake of cutting too close to your ligature, thus causing it to slip off. If this accident occurs during the operation it is not so bad, but when it takes place after the patient has been put to bed it is liable to prove fatal. I now remove the uterus with its attached appendages. As the parts retract the hemorrhage is quite free and seems to be coming from all parts of both broad ligaments without any arterial spurting. It is evident that it will be unsafe to depend on the ligatures, so I shall bring each ligament down by catching its several parts with forceps and then place the large hysterectomy clamps behind them. This is not easy to accomplish, as the upper parts of the structure have retracted high in the pelvis. I now succeed in bringing it altogether on this side, and, taking the clamp, thrust it up outside of the forceps, one jaw being anterior and one posterior to the ligament. Now, feeling carefully with my finger so that I am sure I have not grasped a loop of intestines (this is an accident especially to be guarded against), I close them as tightly as possible. The forceps will now be removed and the opposite side treated in a similar manner. The clamps have caused the hemorrhage to become much less, and what oozing there is will be readily controlled by the

dressings which will now be applied. As you see, I am packing the vagina rather firmly with iodoform gauze, and you notice that I pack between the clamps and use long thick strips that reach from the upper end of the forceps to the outer parts and are laid one over the other. Placed in this manner they better serve the purpose of drainage than if placed in irregular masses, besides allowing later of more ready removal. As the urine must be drawn for several days, I am careful to separate the packing below the urethra and place against the meatus this piece of gauze smeared with carbolized vaseline. This protects the meatus and can be renewed after the water is drawn each time. The handles of the clamps will be steadied by this mass of gauze that I now pack around them, and all held snugly in place by the T-bandage.

The experience with the ligatures in this case, gentlemen, will teach you the advisability of using that method which is the best adapted to each individual case. If I had given up the ligature and applied the clamp as soon as I observed the great thickness of the uterine attachments, considerable time would have been saved.

The uterus, you will observe, is about three times the normal size, the cervix much enlarged and disorganized by the disease, while the tubes and ovaries appear to be healthy. If you will examine the raw surface on each side of the uterus where the broad ligaments were cut away, you will see a few slightly enlarged lymphatics, which may indicate the beginning of infection of the glandular system, and tends to make our prognosis a little less favorable. I will now open the uterus, and as I do so you can see that the mucous membrane towards the fundus is much thickened and inflamed, and as it is passed around you can also see a small growth, as large as a pea, just back of it. This is ocular evidence of the presence of the disease in this part of the organ, and illustrates to you the necessity of removing the whole uterus in cases like this, where the disease is only visible at the cervix, instead of simply amputating the cervix, as is often advocated in such cases. In cancer of the uterus wherever located, the only proper operation is the one that you have just witnessed,—that of total extirpation.

[NOTE.—Patient recovered and left the hospital eighteen days after operation.]

CURETTAGE OF THE UTERUS.

The next case I show you is one simply requiring curettage of the uterus. The patient is very anæmic, as she has been having frequent hemorrhages for over a year and these have been becoming more and more frequent. She menstruates now about every two weeks, and some-

times oftener. The cause of the trouble in this case is somewhat obscure, although her history indicates a six-weeks' abortion as the starting-point. A frequent cause of this condition is abortion, in which some of the contents of the uterus have been retained. The uterus during the early months of gestation is weak and its muscular structure inadequate to expel the contents, consequently more or less of the placental tissue is often retained, and this produces a chronic inflammation of the uterine endometrium, resulting in granulations forming on the mucous membrane,—the condition present in this case. This irritation in time causes subinvolution of the uterus. The organ is large, heavy and spongy, and in that condition that Thomas designates as hyperplasia,—an increased growth of the natural tissues of the uterus. In this connection it is interesting to consider the subject of the removal of the remains of abortion or miscarriage. You should always be certain that the contents of the uterus are entirely expelled in such cases. In cases of abortion with severe hemorrhage and with very little or no dilatation of the os, the old-fashioned way is to tampon. You fill up the vagina and pack it as tightly as possible, and for the pain caused by this procedure you administer opiates. If you plug up the canal thoroughly you will probably find most of the contents of the uterus in the vagina the next morning; but if you have not tamponed very well, you will soon find that the bleeding will be as bad as ever and you will have to put in more plugs; and when you leave the patient it will be with a feeling of dissatisfaction and the fear of recurring hemorrhage haunting you. It is still more unsatisfactory to the patient than to yourself, because she is suffering intensely all the time the tampons are distending the vagina, unless kept fully under opiates. If you succeed, you have yet a week or ten days more of treatment to get her over the shock caused by your manipulation, besides the condition caused by the opiates she has taken.

You should not, of course, remove the contents of the uterus until you know an abortion is inevitable, or that the hemorrhage is dangerous to life. When you have severe hemorrhage in such a case, you do not need to do all this packing. You place the woman immediately in the lithotomy position, and without giving an anæsthetic, and requiring no assistant, use the instrument which I now show you.

The jaws of the forceps are curved, as well as the shanks, allowing of easy introduction in cases of flexion of the uterus. The instrument, being small, can be readily introduced through an os that is but slightly dilated, and the shanks being crossed permits of the wide opening of the jaws when within the uterine cavity without further dilatation of

the os. You introduce it as you would a sound, first passing the finger up to the os and guiding the instrument along it. If you are at all dexterous you need cause your patient no pain whatever. If there is a retroflexion, you introduce the instrument, turn it, and run it into the uterus towards the cul-de-sac, closed with the catch. You should not

FIG. 1.



Longyear's abortion forceps.

expect to remove the secundines *en masse*, but by fragmentation. I wish you to bear in mind this fact, that this is an instrument to be used for removing the remains of abortion by *fragmentation*. Occasionally, where the placenta is entire, lying loose in the uterus, and the os well dilated, you can grasp it and remove it entire; but these conditions are rarely present. You cannot often expect to do this in abortions of from one to two or three months. You introduce it closed, then open it to its fullest extent, and press it against the uterine surface; close it gently—not locking it—and keep going around the uterus in this way until you have doubly curetted, as it were, the whole surface. Remove the instrument each time you feel anything in its grasp, having a jar or vessel before you in which to place the material. After removing in this manner every vestige of foreign material from the uterine cavity, you should complete the operation by washing out the uterus and vagina with a hot antiseptic solution. I am accustomed to use a 1 to 5000 corrosive sublimate solution. When you have thus treated your patient according to the principles of correct surgery, you may feel certain that she is not going to have septicæmia or subinvolution, and that a speedy recovery will ensue. Thus, you do not need to subject your patient to the pain and discomfort of the vaginal tampon, with its attendant long wait for something to happen; but you step in and, neatly and expeditiously, assist nature to do that which she is not yet prepared to do.

Some doctors will tell you that they think it dangerous to introduce instruments into the uterus at this time. It depends entirely on *how* they are introduced. You can safely use instruments made for this purpose, if ordinary care and dexterity are observed.

Before deciding to curette the uterus you should satisfy yourself that there is no fixation of the organ, as this usually indicates disease of the Fallopian tubes, and the manipulation is liable to rupture a pus tube, if one be present, and you will have a dangerous peritonitis resulting. You will remember some other cases of this kind that I have had here, in which I have urged you to be careful in thoroughly examining the parts for malignant disease. We will preserve some of the material to be removed and submit it to the pathologist. You doubtless remember the case on which I operated some two months ago for laceration of the cervix, and at the same time curetted and discovered malignant disease. In that case I removed the uterus before healing had taken place from the first operation. The woman had to work for a living, and the probability was that she would not come back again; so I did the operation, and she made a perfect recovery and is now earning her living.

In any case where you expect to give an anæsthetic for operation, I do not care how simple the case may be, the urine should always be examined. In this case, while suspecting no renal trouble, the formality of examination has been observed. The report, while showing some slight abnormalities, indicates no organic disease. It is as follows: Color, pale yellow; acid reaction; specific gravity 1020; albumin and sugar absent; sediment present, consisting of urates, squamous epithelium, and bacteria.

I now disinfect the uterine cavity with this bichloride solution, using the small intra-uterine syringe. I shall first use this small Nelson dilator, and when sufficient room is obtained to admit of its introduction, finish with the large and powerful Goodell dilator. I now insert the large instrument, and, opening it until firm resistance is felt, fix the set-screw and wait a few minutes for the muscular tension to relax. In dilating you must not try to do so too rapidly, as there is danger of rupturing the uterus by so doing. Go slowly, and give the muscles time to relax with each setting of the screw. I was making this operation some months ago, when, in dilating a cicatricial cervix, it gave way, making a rent up into the abdominal cavity. This accident will occasionally occur and should be guarded against, although if your instruments and hands are perfectly aseptic and the endometrium has been properly disinfected, it is not likely that harm will result from it. The patient referred to had a free watery discharge for three days, but made a good recovery.

Large dilatation is not necessary here, as only sufficient room is required to allow of the easy passage of the curette. I now have three-

quarters of an inch, which is ample. I now introduce the curette, following carefully the curve of the uterus until it reaches the fundus, and scrape downward with it. Apply force only when you draw the instrument towards you, and you can do no damage. The sensation imparted to your fingers, as the curette passes over the surface of the uterus, should tell you something. If it feels soft and yielding it is not unnatural; but if the surface feels hard and imparts a grating sound, you may know that this is unnatural and is suspicious of either a submucous fibroid or malignant disease. In the case I referred to where I curetted and then suspected malignant disease, which was subsequently verified by the microscope, one part of the uterus was so hard and fibrous that many of you heard the curette in passing over it. I find no such condition here. In scraping the concave surface of a flexed uterus you pass in the curette in this way [illustrating], following the curve of the organ, and do not turn it until you get to the top of the uterus, then turn it gently and scrape down with it. In using the sharp curette it is not safe to turn the instrument when the uterus is flexed until you know you are well beyond the flexion, as it might be easily thrust through the uterine wall.

I shall now thoroughly wash out the uterus with this bichloride solution, disinfecting and clearing it of the débris, and then inject a few minims of a glycerin solution of chloride of zinc and carbolic acid—twenty grains of each to the ounce.

I introduce this silver tube, which is a self-retaining stem pessary, for drainage. It is made to retain itself by the little branches which cross each other at the lower opening of the tube, which are closed by the insertion of the staff when ready for use, and released on its removal, when the stem is placed in position. This little instrument is useful in flexions, and, handled with the staff, is very easy of introduction. When you wish to remove it it can be grasped with dressing forceps and pulled out, the branches closing into the fenestræ in the tube (which openings permit drainage); it comes out without any difficulty. I consider this method of draining the uterus far superior to that of packing with iodoform gauze, which is advo-

FIG. 2.



Longyear's stem pessary.

cated by some gynæcologists. It allows of a free flow without the irritation of distention caused by the gauze. The gauze allows only fluids to percolate through it, retaining all the débris and clots. The gauze causes uterine contractions, so that until you remove it your patient is about as comfortable as though she were having a miscarriage. I have tried the gauze packing, but the irritation almost invariably caused a rise of temperature, and occasionally an inflammation of the adnexa, that gave me considerable anxiety. The tube has none of these objections, causing no pain, and can be retained any length of time if occasionally removed and cleansed.

The after-treatment of this case consists of rest in bed one week, the use of hot douches once or twice a day, to relieve congestion, removal and replacing of tube, with cleansing of uterine cavity every third day for ten or twelve days.

LAPAROTOMY FOR OVARIAN TUMOR; VAGINAL HYSTERECTOMY FOR MALIGNANT DISEASE OF THE UTERUS; FIBROIDS OF THE UTERUS.

CLINICAL LECTURE DELIVERED AT HARPER HOSPITAL.

BY J. HENRY CARSTENS, M.D.,

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Gynecologist to Harper Hospital, etc.**

LAPAROTOMY FOR OVARIAN TUMOR.

GENTLEMEN,—This patient is thirty-four years of age, the mother of three children,—one aged nine years, the others prematurely born, the last something over four years ago. The first childbirth was severe and followed by uterine trouble, the patient being confined to her bed for a long time, and she has been ailing ever since. She came to me for the purpose of having a lacerated cervix sewed up, and for retroversion of the uterus; but I found that, although there was a laceration of the cervix, it had granulated over; there was no raw surface and no direct evidence that her trouble had anything to do with the cervix. There was no necessity of operating upon the cervix itself; in fact, an operation would have been dangerous, as I found a tumor back of the uterus which might have contained pus or other irritating fluid, and would very likely have burst during the process of sewing up the cervix; hence, I immediately told the lady that that which was supposed to be a retroverted uterus was really a tumor back of the uterus, between that viscus and the rectum, probably of ovarian origin, or possibly it might be a pus tube. She has been treated for uterine trouble, and more particularly for laceration of the cervix, off and on for eight years. I want you to take notice that I use very few instruments. I simply have one pair of catch forceps, a needle, a knife, and a pair of scissors, and I am able to perform the whole operation myself, my assistant simply holding the tumor up so that I can ligate the pedicle, and the nurse hands me the sponges. Thus, you lessen the danger of septic infection virtually to

the operator. The fewer the instruments the fewer fingers, and the fewer ligatures the less sepsis.

You observe that I make my incision in the median line, going through the skin, fat, and subcutaneous cellular tissue, and lift up the peritoneum with the finger so as to get into the abdominal cavity. I now enlarge my incision for the purpose of removing the Fallopian tube and tumor intact, if it is possible for me to do so. I may rupture it in my effort to take it out whole; and it occasionally occurs. I find it is bound down by adhesions which I shall have to break up. You see it comes out very beautifully. I transfix it with a needle containing kangaroo tendon, and my assistant, Dr. Walker, will hold the growth to one side while I ligate it. I have made my ligature as short as possible. Having disposed of this tumor (the right one), we will turn our attention to the other side. I find the left ovary also adherent and diseased. I transfix it also with a needle, and while my assistant is holding it I simply ligate it, first on one side and then on the other, and I pull it up and see that I have removed all of the diseased tube. I have left no part of it behind. As no blood or fluid has entered the abdominal cavity, we do not need to wash it out. We now take up the layers of peritoneum and sew it up, close the abdominal cavity, and the operation is virtually ended—just taking nine minutes. Having sewed up the peritoneum I now proceed to sew the rectus together, carefully bringing it into apposition; then I take the tendinous insertion of the oblique muscles, bring them together accurately as near as possible as they were before, and, if our operation has been perfectly aseptic, complete union will take place. I now catch a little of the fat and subcutaneous cellular tissue and bring them together with a few stitches, thus reduce the size of the wound, and also follow the course laid out, uniting layer after layer of the same tissue just as it was before the operation. I now proceed to sew the skin together by catching it just at the inner edge, one side after the other, according to the method devised by Dr. Marcy, of Boston. You can see how accurately and beautifully we bring the skin together. This method gives us an absolutely buried suture; no part of it being exposed to the air, consequently you cannot have post-operative infection. Having sewed the skin together, you can see how nice and perfect the apposition is; and if there has been no oversight in the details of our aseptic and antiseptic precautions, it ought to heal up.

The patient was carefully prepared for the operation. Her bowels had been thoroughly cleansed so as to remove all danger from infection. She was given a strong cathartic last night as a preliminary to the

operation this morning, and was douched. She was given an antiseptic bath, clean and new clothing put on which has been perfectly sterilized,—in short, all the usual aseptic and antiseptic precautions have been taken. Just before the operation we again carefully washed the abdomen with soap and water, using a very stiff brush, rinsed it, then washed it with a bichloride solution and then with ether.

Having completed the operation, I shall again wash the abdominal wall and the wound with ether, so that if any microbes have gained entrance into the edges around the skin by any peculiar combination of circumstances, we can destroy them with ether before we apply our dressing, which consists of sterilized gauze moistened in a bichloride solution 1 to 1000, with cotton batting and a bandage over it, fixed by two perineal straps. This dressing is not looked at nor touched for ten days, unless unusual indications present themselves. We simply look at the chart to see if there is any elevation of temperature; but usually we rarely remove the first dressing until eight or ten days have elapsed, when it is taken off, and mild, soothing applications, such as carbolized vaseline, and a plain bandage are applied. On the eleventh or twelfth day we allow the patient to sit up, gradually walk around the room, and usually about the fourteenth day she is permitted to walk around the halls of the hospital. These patients often go home on the thirteenth or fourteenth day, and always on the fifteenth or sixteenth day, after the operation. This may seem unusually quick to many of you, but it is not; and it is probably due to the method we resort to in sewing up the abdominal wound. You notice I have used only three ligatures,—two for the pedicles, and one continuous ligature about eighteen inches long with which I stitched the whole abdominal wound. The ligature I have used in this operation is kangaroo tendon, which is absolutely aseptic and non-irritating, never causes abdominal fistula, and with it you can bring the abdominal wound together nearly as perfectly and accurately as it was before. By having perfect union, the wound becomes so strong that the patient at the end of twelve or fourteen days can walk about without any danger. This is a great advantage in using the kangaroo tendon and in sewing up layer by layer.

As there was a depression in the centre of this growth, I was inclined to think there were two tumors back of the uterus.

The case beautifully illustrates the danger of operating for lacerated cervix, which is considered a very simple operation,—and so it is ordinarily. But when you do not make a careful diagnosis and exclude pus-tubes, hydrosalpinx, and other tumors, you are liable to produce mischief, as the pulling down of the uterus and the rough handling

necessary to do the operation are very liable to cause rupture of the tubes or of the growth; and if the latter contains irritating fluid, a fatal peritonitis is apt to follow.

In examining the specimen I find that the left ovary contains a hard substance, which seems to be a deposit of lime; and only by careful microscopical examination can we tell whether it is simply lime or a bony growth, which sometimes occurs in these cases.

VAGINAL HYSTERECTOMY FOR MALIGNANT DISEASE OF THE UTERUS.

The next patient I present to you is thirty-eight years of age, the mother of four children, who had never been sick, so far as she remembers, until the 1st of December, 1893, when there was an ichorous discharge from the vagina accompanied by profuse hemorrhage. This has continued to increase rapidly in severity until now, when even a digital examination will cause profuse hemorrhage. The discharge is continuous. She has lost flesh rapidly, and an examination reveals a growth as large as a goose-egg springing from the cervix, almost filling the vagina. It is soft to the feel. The diagnosis is a cauliflower excrescence or cancer.

The etiology of cancer is one of great importance, on account of the frequency with which cancer occurs. It occurs more often in the uterus and cervix than in any other part of the body. The question arises, Is cancer of local or constitutional origin? If it were constitutional in its origin, naturally it would occur in different parts of the body with possibly the same frequency; but its more frequent occurrence in the uterus and cervix than in the stomach and breast leads us to think that it is not constitutional, but local. There is no question in my mind that it is originally of local origin; hence, being of local origin, there is no doubt of our ability to extirpate it thoroughly, if we get the case early enough.

What is cancer? Some writers say that it is of parasitic or bacterial origin; that the disease is due to a bacillus. This view is entertained and advocated by a great many practitioners. Some claim to have isolated the bacillus that is the cause of the disease; while others have found changes in the cell-structure that were not at all bacteria, but changed cells. It is very difficult to isolate the special bacillus that causes cancer. In a case of advanced cancerous disease, a great many kinds of bacilli are found in the tissues. Here you notice a large growth attached to the uterus. It seems to be hard and immovable, and I doubt whether we can entirely remove it, because of its having

so extensively involved the surrounding tissues. An operation will do very little good in this case, as there will certainly be a recurrence of the disease. In these cases the question always arises, Will the cancer return? It will in the advanced cases of the disease. The cancer-cells travel along the course of the lymph channels involving the lymphatics, and, finally, they may be deposited in the broad ligament, the liver, brain, or neck; and, although the surgeon may have removed the original cancerous area, the secondary deposit is carried to a great distance, and we are unable to tell when secondary infection has taken place. If the broad ligament and lymphatics seem to be free from the disease and we find the uterus is perfectly movable, we are justified in operating. If we operate on a case where the broad ligament and lymphatics are already involved, we are unable to remove the disease; and what is the result? As the result of the operation directly, and of more or less infection coming on, there will be irritation set up, absorption, and great activity of the lymphatics; and if there is any implantation of cancerous cells, naturally they will grow more rapidly than if the patient was not operated upon. There is no question that in some of these cases an operation hastens the growth of a malignant tumor; while, if we let the patient alone, there will be a slower growth and she may live longer. If you operate and recurrence takes place in a short time, you bring surgery into bad repute among the public. Some of you may say, What is the use of operating for cancer, anyhow? It always returns. We accomplish absolutely nothing by surgical interference. The disease develops just as fast, or faster, than it otherwise would. Let me say to you that I make it a rule that if I am not firmly convinced I can remove every particle of the disease, and that it is a case that will not recur, I do not perform a radical operation. In such cases I simply do a palliative one, and have the patient fully understand it. Notice the cancerous masses I am removing in this case. There is another source of secondary infection, and that is, infection can take place by having the diseased tissue come in contact with the raw surfaces which you make with your knife or scissors; *ergo*, we must be sure to thoroughly disinfect the diseased mass, so that infection from that source cannot take place.

A word again as to the bacteriological origin of the disease and its infectiousness. If it is infectious, it is a very serious question. Experiments have been made on the lower animals with a view to proving the infectiousness of cancer, and it is claimed that some of them have been successful in this regard; still, as a rule, they are not. However, if the disease is infectious, and during the act of operation you should

cut yourself, you are liable to be infected with cancer germs. They may become implanted on a raw surface, and you will eventually become infected with the disease. This is not at all improbable. Most operators have at some time or another pricked themselves in removing cancerous masses without infecting themselves, and still it does not show that we are not liable to become infected with the disease. There must be borne in mind in dealing with this disease two important factors,—the predisposing and the exciting. If we have at any time a predisposition to disease and become infected, it behooves us to be on our guard in this class of cases.

The operation in this case will only give temporary relief. You notice that I am now removing a conical piece of the diseased mass from the cervix, and I wish you to observe the broken-down cancerous tissue. According to the history as given by the patient, the disease is only of three months' duration. I have no doubt it has been growing for a long time, yet has manifested no symptoms until two or three months ago. I shall now remove as much of the cancerous tissue as I can, using a curette to do so; and you notice that the hemorrhage is greatly diminished by getting rid of the diseased mass. What do we accomplish by this operation? We accomplish a great deal in the way of relief. The discharge and hemorrhage will be stopped, as well as the septic condition; and for a while the woman will gain in strength, her life will be prolonged, and she will be more comfortable. The diseased part has now been removed down to healthy tissue, and in order to remove the last vestige of disease we make use of caustics. We apply to the diseased area a solution of chloride of zinc on absorbent cotton. In using chloride of zinc in these cases, it is liable to run along the walls of the vagina, cauterizing everything, even the vulva. In order to overcome that, the late Marion Sims suggested packing the vaginal cavity with tampons containing a saturated solution of bicarbonate of soda. I used to do that before the days of vaginal hysterectomy, but I have since modified the treatment by using dry absorbent cotton, which catches the chloride-of-zinc solution, the solution coming in contact with the mucous membrane and the upper part of the vagina, and not the vulva, where it is most painful. So we pack the vagina in that way. You notice that I use one string; I tie a knot in it so that we know it is the last tampon we put in, and consequently it is the first one to be removed. This is left in for forty-eight hours.

Formerly in this operation, on account of the fear of hemorrhage, persulphate of iron was used in connection with the tampon, allowed to remain for twenty-four hours, then removed and chloride of zinc

used. I found this perfectly useless. Chloride of zinc is as good a styptic as persulphate of iron ; in fact, if the diseased tissues are thoroughly removed and the surgeon gets down to healthy tissue, unless he severs an artery very little hemorrhage will result. Chloride of zinc will eat into the tissues at least one-sixteenth of an inch and destroy them. If there should be any cancerous germs in the part to which it is applied, it will kill them. A very large slough will form in the course of eight or ten days, leaving behind a clear, raw granulation surface which contracts and is covered by epithelium, and the woman is to all intents and purposes well. She will not remain so. We cannot tell how long life will be prolonged. I have known a recurrence of the disease to take place in the course of three months in some of these cases, and in others it did not recur until two years after operation, and the patients during that time were perfectly well. When we get these cases early, we should always perform vaginal hysterectomy. It seems to me in the early removal of cancer we get most brilliant results by this operation. The disease is limited to the uterus for a longer time than to any other part of the body. If we take cancer of the breast, for example, the lymphatics in the arm are very soon involved ; but if you get a case of uterine cancer early and remove every vestige of the disease, recurrence will not take place. I can show you cases that were operated on five and six years ago, and the women are perfectly well ; and I am firmly convinced that recurrence will not take place in those cases, because of early operative interference. But when you get a case like this, vaginal hysterectomy is of little or no avail. In the first place, the patient is very liable to die as a result of the operation, on account of the great difficulty in getting the uterus down and clamping the broad ligament if you use a clamp, or ligating it if you use a ligature ; and in undergoing such a serious operation the compensation she will get, even if she gets over it, is comparatively *nil*. Recurrence will take place just as rapidly. In the case before you I have removed the whole cervix up to the internal os. All of the diseased area has been apparently removed. The patient will be just as well now as after vaginal hysterectomy, and recurrence will take place just as fast as if the other operation were done, and still no faster. The point I wish to impress upon your minds is the necessity of early diagnosis of cancer. Any old woman could have diagnosed this case, but if you can diagnose a case in its incipency you will be able to thoroughly eradicate the disease ; consequently you must familiarize yourselves with the symptoms of cancer. There are cases, however, that you cannot diagnose with the naked eye, and you must have the assistance of the micro-

scope. I have repeatedly been called upon to operate for lacerated cervix, but from the general appearance of the parts I have become suspicious that there was present malignant disease; and in order to remove every vestige of doubt, I took some of the diseased tissue, had it examined under the microscope, and in many instances my diagnoses were verified. Then, of course, by resorting to vaginal hysterectomy in the early stage of the disease, I secured brilliant results. When you have a case of lacerated cervix that has been raw for a long time, manifesting no tendency to heal up readily, which easily bleeds, accompanied by more or less discharge from the uterus of an ichorous, irritating nature, sometimes bad smelling, you must be suspicious, especially if the woman is about forty years of age. Do not understand me to say that younger woman may not have cancer, for I have operated on women for cancer of the uterus whose ages ranged from twenty-seven to thirty years, but the disease is not so liable to occur before thirty-five years of age, and it generally occurs between forty and fifty. Take a woman who has had long-continued trouble with the womb, the so-called ulcer, who has been treated off and on for years, if she is thirty-five or forty years of age and has a lacerated cervix, or a spot that easily bleeds, accompanied by a discharge, you want to be suspicious; and if you desire to do justice to your patient, you will not stop there. You will remove some of the diseased tissue and subject it to a thorough microscopical examination. You can do that by taking a sharp curette and scraping away a little of the diseased tissue. You place it on a piece of paper, then subsequently put it in alcohol, and examine it at your leisure, or send it to some expert microscopist. If you have microscopic evidences of cancer, you should perform vaginal hysterectomy. It is no use to sew up the lacerated cervix, but you should absolutely remove every vestige of the malignant disease,—the whole uterus; then you get most brilliant results, and only then.

FIBROIDS OF THE UTERUS.

Dr. Howell, of Tecumseh, has kindly brought this patient for operation. She is a colored woman, forty-eight years of age, the mother of four children, the youngest of whom is now seventeen years old. Her general health has always been good until about a year ago. Menstruation has been regular and normal, never profuse, and is regular up to the present day. About a year ago she noticed a tumor in the abdomen which has increased in size and become nodular, and is causing her a great deal of distress at present. You can see that her abdomen is quite large and irregular in shape, and contains a number of hard

tumors which are more or less connected,—a plain case of multi-fibroids of the uterus.

As she has already received general constitutional treatment, and as in this variety of fibroid tumors electricity certainly has no effect, the only thing we can do is to operate on her; and as she has been thoroughly prepared by purging and aseptic baths and douches, and having her thoroughly under the influence of chloroform, we shall proceed to operate. The exact operation I shall perform I have not decided. That can be settled as we proceed. We may make a total extirpation, we may use the clamp, or we may use the ligature, and drop the stump,—i.e., the cervix,—and sew up the abdominal wound. As the growth is very large, I shall immediately make an incision from the umbilicus to the symphysis, which is six inches. I now try to remove the growth, and, although the incision stretches, it is still quite difficult. But I get out one growth, and by twisting it around I get out another growth, and thus gradually get the whole mass outside the abdominal cavity. I would have, perhaps, less trouble if I had enlarged the incision, but I do not like to go above the umbilicus if I can help it, as we often get a hernia at this place after the operation. Having removed the whole mass, we can here see a very long cervix, and it seems to me an easy case for total removal, leaving in the cervix simply. I now proceed to ligate the ovarian artery on each side, and also the uterine artery, thus absolutely controlling hemorrhage. I cut through the broad ligament on each side up to the uterus, and now have the elongated cervix simply holding the tumor. This is about as thick as my thumb. I make a wedge-shaped incision; the tumor is entirely removed, containing the fundus of the uterus, and ovaries and tubes of both sides. With an ordinary Hagadorn needle I sew the peritoneum over the raw surfaces, sew up this wedge-shaped incision in the cervix in two layers, and now have an absolutely clean abdominal cavity. For fear, however, that some septic material might have escaped from the end of the cervix, I thoroughly wash it out with normal saline solution, and then insert a drainage-tube (which I do not think necessary in this case, still it will do no harm), and we may thus remove fluids from the peritoneal cavity which might be the starting-point of septic infection. Using a Hagadorn needle slightly curved at the end, I sew up the abdominal wound, first, with a running suture, sewing the peritoneum from above downward, and then, running up with the same ligature, bringing together the rectus muscle, and then down again towards the symphysis with a continuing running suture, carefully adjusting the tendinous insertions of the oblique muscles.

Now, with a few stitches I bring the fat together, which, in this case, is very sparse; and then we come to the skin, which is the most important, and must be done most carefully. I make use of a stitch devised by Marcy, catching the skin in the rear about half-way, and then bringing it together. Continuing that way, we have our sutures buried, and still bring the skin carefully into approximation. I use for all my ligatures, inside and outside, in the abdominal wound, the kangaroo tendon; which, if absolutely aseptic, is completely absorbed and the wound is dry and firm in ten or twelve days, so that I allow patients to sit up in ten days, and two weeks after the operation I allow them to go home. Sometimes I keep them a few days longer, but generally, as said, on the tenth day they can get out of bed, by the twelfth day can walk around their room, and on the fourteenth day can walk all over the hospital. Such results cannot be shown when we use the large *en masse* suture, using silk, silver wire, or silkworm gut. The ideal method certainly is the one of bringing into apposition the tissues as they were before the incision was made, bringing together peritoneum to peritoneum, muscle to muscle, fascia to fascia, fat to fat, skin to skin, and when we have union by first intention there is no danger of abdominal hernia.

Ophthalmology.

TREATMENT AND ETIOLOGY OF CONJUNCTIVITIS; HYPEROPIC ASTIGMATISM; IRITIS SYPHILITICA.

DELIVERED AT THE NEW YORK POLYCLINIC.

BY THOMAS R. POOLEY, M.D.,

Professor of Ophthalmology in the New York Polyclinic; Surgeon-in-Chief of the New Amsterdam Eye and Ear Hospital.

GENTLEMEN,—Examining the patient with conjunctivitis, whom we have been treating with nitrate of silver, you notice that there is still some catarrhal secretion. We have been using a one-per-cent. solution of the nitrate of silver, washing it off with water after each application. Where strong solutions are employed salt-water may be used. Many of the younger men in the profession are very fond of diagnosing the form of conjunctival disease by a microscopical examination of the secretion, and so determining whether or not it be contagious. Thus, they tell us that catarrhal forms of conjunctival disease, which are especially prevalent in the spring, and which are endemic, recurring in the same localities season after season, are not contagious. I do not think that we can consider any such examination a safe guide in this respect. My clinical experience in this class of cases is opposed to such a view. I believe that any eye-disease, in which there is a secretion which persists, and is of such a character that it can be disseminated through direct contagion, or by propagation through the air of crowded dormitories, asylums, and hospitals, is likely to infect other cases, and I am almost certain that the same kind of conjunctival inflammation is not always set up; in other words, that a catarrhal conjunctivitis may excite in another case a purulent form of the disease. Hence, I consider these modern teachings dangerous. The man who makes his clinical laws and deductions from peering through the microscope is pretty sure to be led astray, and, although the microscope tells us much that is useful, a man who makes this subject a hobby is, to my mind, an unsafe adviser and practitioner.

Here is a young girl, seventeen years old, who tells us that she cannot see objects at a distance, and that work must be held close to

her eyes. She says that her sight became worse five years ago. I wish you to notice particularly the age at which this change was noticed. She says that it developed just after an attack of scarlet fever, and that her physician said that her poor sight was a result of this disease. I have examined her eyes and there is no evidence of the sequelæ of scarlet fever. It is instructive to analyze an affection of this kind. What can be the condition after a severe illness which would affect the eyes in such a way as to render the vision defective for all these years? In scarlet fever there may be an affection of the kidneys—an acute desquamative nephritis, producing all the symptoms of Bright's disease, and, in some cases, producing an affection of the optic nerve and retina. Had this been the condition in this patient recovery must have occurred long ago, and permanent damage could only have resulted from leaving atrophy of the optic nerve and of the retina in the macular region. Ophthalmoscopic examination has shown that such a condition can be excluded here. Again, she may have had a more common sequela of scarlet fever,—paralysis of accommodation. By paralysis of accommodation we mean that the ability of an eye to adapt itself to near vision by increasing the curvature of the lens, through the action of the ciliary muscle, is lost. If the word "paralysis" is used, it means that the accommodation is *nil*; if the word "paresis" is used, it means that there is a feebleness of the accommodation, but not a total loss of it. If this were the case the patient's pupils would be dilated, and she would see better the farther she removed the object from her eyes; hence, we may exclude paralysis of accommodation; and, moreover, such a condition would hardly last for five years. A still more common way in which the eye is damaged in all exanthematous diseases, especially in scarlet fever, is by an inflammation of the conjunctiva, extending to and affecting the cornea, or by ulcerative processes in the cornea itself, leaving opacities on this membrane. The corneæ here are perfectly clear, and hence this condition can be excluded. We have, then, to look elsewhere for the causes of the defective vision in this girl's case, and in making an examination we may first determine her acuteness of vision, and, in the next place, whether any glasses will improve her vision. If there be a notable decrease in the acuteness of vision there are two forms of error of refraction which we most suspect. In myopia of moderate or high degree there is rarely acute vision for distance; a patient, therefore, with bad vision, especially if she holds the object near, may be myopic. If we examine a patient as to the acuteness of vision, and try respectively convex and concave glasses, and the sight is not improved, we suspect that the patient has

astigmatism. Analyzing our case as far as we can from the history, the probabilities are that one or both of these errors of refraction exist, *i. e.*, simple myopia or astigmatism, either myopic or hyperopic.

Now, we must ascertain what is the nature of the error of refraction. You may divide errors of refraction conveniently into hypermetropia, hyperopic astigmatism, simple and compound, myopia, myopic astigmatism, simple and compound, and mixed astigmatism. By mixed astigmatism we mean that there is astigmatism of both the myopic and hyperopic form, so that the focus for the object may lie in front of the retina for one series of lines and behind it for another. There may also be irregular astigmatism, in which the quality of the astigmatic aberration is such that it cannot be corrected by glasses. This girl is not now under the influence of any mydriatic ; you must be guided in their use by the peculiarity of the individual case. Dr. Roosa, among others, says that he no longer uses atropine, as he can make the examination equally well with Javal's optometer. This new instrument tells you whether there is any astigmatism of the cornea, which, of course, is very little influenced by the atropine. Other observers do not believe that the determination of the refraction can possibly be made without the use of atropine. I believe the truth lies between these two extremes. There are cases where atropine is entirely superfluous, as in persons in middle life coming to you complaining of asthenopia, where you find hypermetropia on examination. The strongest glass such a patient "accepts" indicates the "manifest hypermetropia." If the patient sees best with a convex glass, the strongest glass of this kind with which he can see in the distance is the expression of his manifest hypermetropia, and you can safely prescribe this glass for him. In people of middle age the accommodation is at a minimum, and all hypermetropes exert the accommodation for the distance, and if you prescribe a glass which exactly neutralizes the manifest hypermetropia, you will at once relieve the asthenopia. If the case be quite severe you order the glasses to be worn constantly ; but if a patient comes to you, and you obtain varying results,—they see equally well with either convex or concave glasses up to a certain degree, and from the statement of the patient you find it is impossible to determine what glass will correct the error of refraction,—it may be proper to employ atropine. It is better also in young people with errors of refraction, where the accommodative act is very energetic, the results varying, and the asthenopia considerable.

Now, testing this patient's vision, we find that it is $\frac{7}{8}$ in both eyes. Such vision indicates myopia or some form of astigmatism. We find hypermetropia of $\frac{1}{20}$ in the left eye. We know that the spherical glass

with which we get the best result is a plus $\frac{1}{20}$, and we proceed to see whether if we add a cylindrical glass to that the patient sees better. A plus $\frac{1}{12}$ cylinder with the axis vertical, and a plus $\frac{1}{20}$ spherical give the patient vision of $\frac{2}{3}$. This is very imperfect vision. If correction of the error of refraction does not secure better vision, search for some other cause for the defect; and in these astigmatic cases it frequently happens that this is due to irregular astigmatism, which we cannot correct. As she accepts plus $\frac{1}{4}$ before using a cylinder I should expect that a complete paralysis of this girl's accommodation would show more hypermetropia. If we can bring this patient's vision to $\frac{2}{3}$ we have accomplished a good deal. I may also say that when such a patient wears a suitable glass habitually the acuteness of vision improves. Be careful about conclusions from examinations made with very weak glasses, either spherical or cylindrical. Now, examining the right eye, we find that a $\frac{1}{40}$ spherical with a $\frac{1}{12}$ cylinder, with the axis at ninety degrees, brings the vision to $\frac{2}{3}$. We shall have to postpone for the present the examination of the eye under atropine.

This colored man was presented to this class some time ago as a case of iridocyclitis. At that time there was an inflammation of the iris with a synechia, but when he first came to me with a history of traumatism there was great pain, circumcorneal injection, a moderately dilated pupil, and distinct pain upon pressure over the ciliary body, a few degrees to the inner side of the vertical meridian. This painful point, together with the history of traumatism, and the fact that the ophthalmoscope showed the vitreous humor to be cloudy, caused me to make a diagnosis of iridocyclitis. He was admitted to this hospital and treated accordingly; but after awhile he not only developed a more distinct iritis, but he broke out with a general syphilitic eruption. It is probable that the subsequent severe symptoms were due to the complication of this constitutional disease. Now we find the cyclitis is entirely gone. In specific iritis there is not usually pain on pressure, so that I think the original diagnosis was correct. There is now some circumcorneal injection, showing the usual relapsing form of syphilitic iritis. Just under his chin you will feel an enormously enlarged gland. In colored people specific disease of the eye often assumes a very severe type. The usual mercurial course of treatment must be accompanied by cod-liver oil, and the patient must be placed in good hygienic condition. At present atropine should be instilled into the eye. These relapses in specific iritis nearly always yield to treatment much more easily than the original disease.

DIABETIC AFFECTIONS OF THE EYE.

CLINICAL LECTURE DELIVERED AT THE NEW YORK POST-GRADUATE MEDICAL
SCHOOL AND HOSPITAL.

BY WILLIAM OLIVER MOORE, M.D.,

Professor of Diseases of the Eye and Ear at the New York Post-Graduate Medical
School.

"What says the doctor to my water?
He said, sir, the water itself was a good,
Healthy water, but for the party that owed it,
He might have more diseases than he knew of."

—*King Henry IV., Part II.*

GENTLEMEN,—The patient whom I wish to present to you to-day will answer for a text on diabetic affections of the eye. During the past fifteen years I have seen an unusually large number of diabetic affections of the eye, as compared with the number of such cases reported in the practice of other physicians. This is to be accounted for by my special interest in this class of patients, and my solicitation of diabetics from general practice for ophthalmoscopic examination; thus, I have sought for the cases before they sought the ophthalmic physician. What I may say will contain nothing specially new, but will be rather a report of my own cases, together with a *résumé* of the subject as it is at present understood, with the hope of bringing it more particularly to the attention of the profession.

Celsus was probably the first to clearly describe diabetes, and Galen (131–210 A.D.) first to call it by that name. In 1674 Willis first associated the presence of sugar with the disease,—discovered by the sweet taste of the urine. Other observers succeeded in separating the sugar, and Rollo, in 1787, began the dietetic treatment by withholding vegetable food. It is not a rare disease; it is more common now than formerly. This is probably due to the frequent and more accurate examination of the urine practised at the present time.

**REPORT OF DEATHS FROM DIABETES IN NEW YORK CITY. FROM
THE RECORDS OF THE BOARD OF HEALTH.¹**

	Deaths from Diabetes.		Deaths from all Causes.	
	Total.	Over 15 years.	Total.	Over 15 years.
1878	42	41	27,008	13,174
1879	37	36	28,842	14,075
1880	44	43	31,987	15,684
1881	43	43	38,624	18,250
1882	46	43	37,924	18,264
1883	68	64	34,011	18,451
1884	68	67	35,034	18,184
1885	63	62	35,682	18,787
1886	82	78	37,851	19,566
1887	105	104	38,933	20,808
1888	117	112	40,175	20,841
1889	118	117	39,679	20,710
1890	130	127	40,108	22,331
1891	130	126	43,659	23,506
1892	123	118	44,829	23,671
Total	1216	1171	552,791	285,797

Total deaths in fifteen years from diabetes, twelve hundred and sixteen.

The total deaths from all diseases in fifteen years, five hundred and fifty-two thousand seven hundred and ninety-one; one diabetic in every four hundred and fifty-four deaths.

In 1867-69 the ratio of deaths from diabetes to the total number of deaths was 1 to 1379; in 1867-69 there were forty-nine deaths from diabetes; in 1877-79 there were one hundred and twenty-three deaths, an increase of seventy-four; which is out of proportion to the population increase. It is more common between thirty and forty years of age, but it is seen in early life, although the earlier writers did not so consider. Prout saw, of seven hundred cases of diabetes he had met with, but one instance in a child five years old, and only twelve in young persons between eight and twenty years of age. Hanner has reported the case of an infant twelve months old; G. M. Smith, M.D., reports one twenty-three months old, and W. H. Dean, M.D., of Blanford, Mass., a female fifteen months old. Of the cases treated at the New York Post-Graduate School and Hospital, there were only three in the first ten thousand; in the Vienna Poliklinik, Romberg found the same ratio to exist. I am quite sure the disease is more prevalent in private

¹ I am indebted for the foregoing to Dr. Nagle, registrar of vital statistics.

practice than is usually conceded, and the reason we do not find records of such in hospitals and dispensaries is on account of the carelessness of the examinations and the improper signing of death certificates. There is also a great disproportion between diabetic affections of the eye seen in private and those seen in hospital practice, owing to the loose way of making the diagnosis. I make this sweeping statement from my recent efforts to obtain evidence as to this point in the principal ophthalmic hospitals of this country. The statistics of these institutions are utterly useless. Hirschberg remarks upon the much greater frequency of diabetes in private than in hospital ophthalmic practice, and reports sixteen hundred and thirty-five private patients, and seventeen suffering from diabetes; while of ten thousand at his clinic, three only had sugar in the urine. During the past fifteen years I have examined one hundred diabetic subjects, and of this number twenty-one had ocular affections, as follows:

Amblyopia (without ophthalmoscopic change)	4
Cataract	4
Retinitis glycosurica	5
Hemorrhages and floating bodies in the vitreous	4
Paralysis of accommodation	8
Iritis diabetica	1
Total	21

Dufresne out of one hundred and sixty-two diabetics had twenty with various ocular affections. In 1798, Rollo first called attention to eye-trouble in diabetics, although Blankard, in 1688, mentions a case of blindness in this disease, in which, however, it was subsequently found both the diabetes and the blindness were due to a tumor of the brain. Dr. Prout and Mr. France long since called attention to this association. The most common ocular symptoms usually are: (1) Affections of the muscular system,—paralysis or loss of accommodation, and paralysis of the extrinsic muscles of the eye, usually the abducens. (2) Affections of the vascular system,—in which we observe hemorrhages into the vitreous and also floating bodies, the result of improper nutrition. As a result of the serious changes in the vascular system retinal hemorrhages take place, and are usually associated with hemorrhages in the vitreous. (3) Cataract and amblyopia are found in the later stages of the disease. (4) Iritis and keratitis are rarely seen, but have been admirably described by Leber, by Schirmer, and Wiesinger.

It is quite natural that the first ocular symptoms noticed should occur as the result of general muscular weakness. I have seen many

diabetics with temporary loss of accommodation lasting from a few days to a week, when more than the usual amount of sugar was being voided from the system. This condition would yield readily to proper treatment. The cases where the accommodative disturbance was but transitory I have not counted, and I will relate only one where it remained permanent.

A male, thirty years of age, a lawyer by occupation, first consulted me in 1879, complaining of difficulty in reading. On examination vision equalled $\frac{3}{8}$, and was not improved by glasses. No ophthalmoscopic change was present; the pupils were normal, but accommodation was rather faulty so that newspaper type could be seen only at eighteen inches. Convex glasses (+ 1.25 D) were ordered for reading, and near vision was restored. This patient subsequently had opacities in the vitreous, and died eighteen months later from phthisis superinduced by this diabetic condition.

Next to failure of accommodation, the muscular system shows its weakness in paresis of the abducens or sixth nerve. I have never seen this affection result from diabetes; or, if so seen, it has never been recognized by me. Gutmann recorded in 1883 a case occurring in the person of a physician, fifty-three years of age, who was thought to be suffering from tabes, but he was afterwards found to have diabetes. He was seen by Hirschberg, who found paralysis of the right abducens with normal pupillary reaction and accommodation. The paralysis of the abducens was complete. After a sojourn at Carlsbad for two months the sugar diminished and finally disappeared, and with it the diplopia produced by the affected muscle. The fact that the sixth nerve takes its deep origin from the floor of the fourth ventricle, a portion of the brain usually affected by diabetes, is supposed to be the reason why this particular muscle is paralyzed, as the other ocular muscles get their innervation from other parts more remote from this origin.

Iritis and keratitis from diabetes have been noticed, and Leber and Wiesinger, in 1885, report such cases, most of which occurred in the Göttingen clinic. In 1857, Leudet, in the *Gazette Médicale*, relates the case of a female diabetic, thirty-two years of age, with keratitis of the left eye. This patient, however, had paralysis of the third and fifth nerve on the same side, and at the autopsy a tumor, syphilitic in character, was found in the brain; so that this case was not diabetic but neuro-paralytic in origin. In 1830, Himby speaks of keratitis in connection with diabetes, but his cases were not clearly defined. Panas, Galezowski, Bellouard, Condouris, and others have reported cases of diabetic keratitis, and in almost every instance it has been of the puru-

lent form, and the inflammation of a very passive character. Most of the cases reported terminated in complete leucoma and some in leucoma adherens. From what literature I have had access to it would seem that keratitis from diabetes alone is a rare affection. *Iritis* seems to be more common. The first case reported, so far as I can learn, was in 1863, by Marchal de Calvi, that of a female diabetic, fifty years of age, who had irido-choroiditis, and who died shortly afterwards from the general affection. In 1868, at the American Ophthalmological Society, Dr. H. D. Noyes, of New York, reported a case of retinitis hemorrhagica occurring in a diabetic woman, sixty years of age, who had had some months previous to coming under his observation double iritis, the posterior synechiæ showing themselves at the time of the examination. This case was reported by Noyes for the intra-ocular affection, and only incidentally are the results of the iritis mentioned, he evidently not considering the iritis due to the diabetes. It, however, belongs to that class. Wickersheimer, in 1874, reports a male, thirty-seven years of age, with irido-choroiditis. Galezowski, in 1879 and 1883, relates the case of a male, sixty years of age, with iritis in the left eye and synechiæ in both. For two years the right eye had been the seat of iritis, he having been diabetic since 1866. The left eye improved in three months. Abadie in the same year reports a similar case. Umman, in 1881, reports the case of a priest, sixty-nine years old, with purulent iritis. Condouris reports also a case of a diabetic female having iritis. Leber reports nine cases of iritis with full and complete histories.

I have seen only one case of iritis in a diabetic, and it occurred only two months before the death of the patient. The following is a short synopsis of his history: A male, thirty-five years of age, a laborer, for two years has lost flesh and has grown weaker; March 1, 1885, passing seven pints of urine daily, and eleven grains of sugar to the ounce; never had any trouble with his eyes. Two weeks before seeking advice he first noticed cloudiness of vision in the right eye, followed in six days by failure of vision in the left eye. When seen right vision = $\frac{2}{8}$, and left vision = $\frac{3}{8}$. The right iris was inflamed and there were one or two small synechiæ, showing the aqueous cloudy and a small amount of hypopyon. Slight injection of the eyeball was present, but there was not much pain; the left pupil was clear, though slowly responding to light; circumcorneal injection, slight pain, and no fear of light. Ophthalmoscope showed the media clear. Four days later each eye had hypopyon and more marked signs of iritis. The cornea of the right was slightly cloudy in the outer quadrant. Careful diet was pre-

scribed and the usual local measures for iritis were adopted. The patient recovered with a few adhesions remaining in the right eye, and some in the left eye after an inflammation of six weeks' duration. The cornea remained cloudy up to the time of his death.

I have in a number of diabetics noticed a want of clearness of the vitreous where no opacity could be really determined, and this would vary with the quantity of sugar in the urine. I suppose it is to be explained by the varying condition of the blood in these cases. I would here refer to a case where an actual change could be distinctly seen.

Floating Bodies in the Vitreous and Opacities in the Lens.—The patient was a woman, fifty years of age, who first showed signs of diabetes four years before. About six months before coming under my notice she complained of dimness of vision, with "specks before the eyes." These symptoms gradually increased, and when seen, in 1884, vision was $= \frac{1}{4}$ in each eye; the pupils were normal; the visual field was normal, and the ophthalmoscope showed very many floating bodies in the vitreous, and some small fresh hemorrhages. The appearance was about alike in each eye. The optic nerve and retina were normal. The lens showed a few striæ in the peripheral portion. At this time she could read with correcting glasses for a short time only. Much sugar was being voided. Whenever the quantity of sugar in the urine diminished her vision improved. During the remainder of her life her eyes gave her annoyance, and on two occasions, for a period of five weeks, during which time the diabetic symptoms were more pronounced than usual, vision was so bad that reading could not be indulged in. On these two occasions the opacity of the lens was much increased, and the lens, in fact, generally cloudy. On the disappearance of the severe diabetic symptoms the lens gradually cleared, the floating bodies in the vitreous remaining, but the vision improving so that she could read for a short time.

This case would seem to be one like that reported by Nettleship, where cataract disappeared on the subsidence of the severe diabetic symptoms. The patient died in diabetic coma in December, 1887, with no increase in the opacity of the lenses or vitreous. The patient was the wife of a physician in this city, and was very carefully treated.

Cataract.—Opacity of the crystalline lens has from the very earliest times been considered as one of the most common affections of the eye in diabetes mellitus, and it is only recently that the text-books have spoken of other diabetic affections of the visual apparatus. According to Roberts, it occurs once in forty-five cases, and according to Bouch-

ardat, once in thirty-eight diabetics; while Griesinger, out of two hundred and twenty-five, found cataract to exist in twenty. In St. George's Hospital it occurred once in twenty-eight cases; Frerichs found nineteen out of four hundred, and Graefe asserts that he has found cataract present in one-fourth of all diabetics. In eight hundred and fifty-eight diabetics collected from various sources already reported, together with my own, I find that diabetic cataract occurs once in seventeen persons whose glycogenic function is disturbed. In regard to the relative frequency of diabetic cataract as compared with other forms of lenticular opacity it is difficult to say, as in the various hospitals the diagnosis is not accurately made, the special kind of cataract being rarely recorded. For example, out of eighty-nine hundred and thirty-three cases of soft and hard cataract, non-traumatic and non-congenital, recorded in the principal eye hospitals in New York, Brooklyn, Philadelphia, Boston, and Baltimore, only four were recorded as of diabetic origin, or one diabetic cataract in every two thousand and twenty-three cases of opacities of the lens. I am quite sure that this ratio is far too small. Badal, out of twenty thousand patients with eye-diseases, found fifty-two with diabetic affections of the eye, and of the latter number thirteen were cataract. It occurs in advanced cases, yet it also happens that attention is first directed to the diabetes by the disturbance of vision, more especially by the rapid formation of the cataract. As a rule, both eyes are attacked, though the maturing is not usually equal.

The origin of cataract in diabetes was formerly attributed to the withdrawal of water, from the experiments made by Kunde in 1856, who produced an opacity of the lens in frogs by desiccation and the introduction of substances under the skin having a strong affinity for water, such as salt and sugar. These experiments were afterwards confirmed by Weir Mitchell and others. Von Graefe, on the contrary, pointed out that such lenses (diabetic) did not, like those of frogs, lose their opacity by being laid in water, and that they did not, like the latter, show any formation of vacuoles in their interior. The non-appearance of cataract in diabetes insipidus also tells against its origin in the mere deprivation of water. In view of the fact of spontaneous disappearance of cataract in several cases on improvement of the diabetic symptoms and decrease in the sugar voided, as reported by Seegen, Nettleship, and others, and as seen in the case to which we shall refer, it would seem more probable that the abnormal constitution of the blood, its saccharinity, as well as that of the other fluids, favors the formation of cataract. Mauthner says, "We can hardly deny that a man at middle life with double cataract is suffering from marasmus;

indeed, I have admitted this in diabetic cataract,—that is, in cataract which has appeared rapidly from the diabetic origin of large excessive watery extraction or a severe disturbance of nutrition, but not from the presence of sugar in any internal structure." Schweigger says, "I include under diabetic cataract only cases where deeply-seated cataract has developed rapidly in young individuals who are in an advanced stage of acknowledged diabetes." Sugar has been detected in the lens and in the aqueous and vitreous humors, as shown by Liebreich, Knapp, and Lohmeyer. In my own experience, I have seen but four diabetic cataracts, or four per cent. of the diabetics examined.

I will refer to a case which I have recently seen,—a male, sixty-one years of age, a merchant, seen in private practice in 1885, with a history of failing vision for the past four months, associated with an increase in the quantity of urine passed, the latter containing much sugar and having a specific gravity of 1040. There was no gout or rheumatism, or anything of importance in his family history. Examination of the eyes revealed in the right incipient cataract with striæ, in many places having the usual appearance of senile cataract; in the left eye the lesion looked more like the soft variety, and was more opalescent. The fundus was normal; right vision was $\frac{3}{8}$, left vision $\frac{3}{8}$. This patient was placed on careful diet, and iodide of potassium prescribed. This treatment was kept up seven months faithfully, with the result of improving very much the general condition and with some effect on the opacities in the lenses. One year after the first examination right vision was $\frac{20}{80}$, left vision $\frac{3}{8}$. The patient died of diabetic coma three years after the first appearance of the general disease, and without any operation on the cataract.

Besides these four cases which I have seen, I know of one in the practice of the late Dr. Loring, where he extracted a cataract in a diabetic subject, with very excellent result and without any reaction. I know also of one case in the practice of Dr. Thomas R. Pooley. We are to make a distinction between cataracts occurring in diabetic subjects from ordinary senile changes, and those coming from loss of nutrition produced by disturbance of the glycogenic function; for we may, I think, extract with freedom those lenses occurring in old people having sugar in the urine, whereas in young diabetics we may have serious inflammation and necrosis of the cornea follow operations for the removal of cataract. I should not hesitate to operate on any case of cataract in a diabetic, after a few weeks' treatment of the general disease by proper remedies; and I think the poor results previously reported were due to the infection of the parts, which, by their lowered

vitality, had less power to resist the germs, and that if proper antiseptics had been employed better results would have been obtained. The spontaneous disappearance of cataract, as above mentioned, gives us a point in the treatment,—to try by all means to diminish the quantity of urine voided, and especially to look for syphilis as the cause of the diabetes. When it is found, iodide of potassium will prove to have a most excellent effect. The use of Carlsbad water is said to have caused diabetic cataract to disappear, or the opacity to greatly diminish.

Diabetic Retinitis.—We next come to the deep-seated affections of the eye in which the long-continued derangement of the glycogenic function causes changes in the vascular system generally, and where hemorrhages are prone to occur in any part of the body,—as the brain, retina, or spleen,—due to the diabetic toxine. According to Artigalas, whatever the symptoms associated with retinal hemorrhages, they are always dependent upon fibroid degeneration of the arteries; and, if the degenerative process continues, cerebral hemorrhage is very apt to follow. Mauthner, in his work on the ophthalmoscope, says Jaeger found retinitis in persons with diabetes mellitus; others have since then declared it possible, and by Martin and Galezowski it is positively asserted. Jaeger's case was that of a male twenty-two years of age. Bouchut, Desmarres, Martin, and others report similar cases, and seem to be unable to distinguish it from albuminuric retinitis. Noyes, at the American Ophthalmological Society, in 1868, reported the case of a woman, diabetic for several years, and who in the previous year had had a double iritis resulting in posterior synechia. When seen, in the retina of each eye were found specks like fatty degeneration with a few hemorrhages. In the right eye the vitreous was noticed to be hazy. He says the appearances are absolutely identical with those he had often seen in cases of chronic renal disease, and the case was at first so considered. Minute and careful records were kept of the urine passed and of the tests made, and sugar only, without albumen, discovered. In concluding the history of the case, he says, "Pure glycosuria is capable of causing retinitis, and that the ophthalmoscopic appearances cannot be distinguished from those which belong to albuminuric retinitis." I am of the opinion that retinitis occurring in diabetes before the kidneys are diseased can be diagnosticated as such, and that it has distinctive characters; that, however, when the kidneys are diseased, as so frequently happens in the latter part of diabetes, we get a mixed form of retinitis which is indeed difficult to determine from the ophthalmoscopic picture. Lagrange gives the following points in the differentiation of retinitis of Bright's disease from that of diabetes:

DIABETIC RETINITIS.

1. Tendency to marked atrophy of the optic nerve.
2. Hemorrhages multiple, disseminated and alone.
3. Extensive alterations of the fundus throughout.
4. Hemorrhages non-continuing throughout.
5. Exudations small in amount and scattered.
6. Changes frequently.

ALBUMINURIC RETINITIS.

1. Less marked, and the nerve not becoming entirely atrophic.
2. Hemorrhages equally multiple, but mostly in the posterior part of the fundus.
3. Mostly in the macula lutea and optic nerve.
4. Remaining throughout.
5. Exudations white, fatty in character, with much infiltration of the retina and nerve.
6. Remains a long time the same.

One symptom is, however, more characteristic,—viz., hemorrhages into the vitreous are common in diabetic retinitis, not so from retinitis with kidney lesions. This is to me an important point in the diagnosis, and is a clinical fact which has been noticed by several observers. Why they should occur in this affection and not in Bright's disease is probably due to the more marked vascular changes in diabetes. As regards the frequency of retinal affection, Badal, in fifty-two diabetic affections of the eye, found retinitis seventeen times; and in our twenty-one diabetic affections of the eye, five were retinitis with hemorrhages, or somewhat over one-fourth of the cases. In the eye infirmaries of this country for a space of ten years I found no recorded cases of the disease. That it does occur is certain, but accurate records are not made.

I find that it is time to close this lecture, so we shall continue this subject in our next, beginning with a consideration of the patient we have here to-day.

Laryngology, Pharyngology, Rhinology, and Otology.

THE ETIOLOGY, PATHOLOGY, AND TREATMENT OF ACUTE AND CHRONIC OTITIS MEDIA PURU- LENTA.

CLINICAL LECTURE DELIVERED AT THE MANCHESTER INSTITUTION FOR DISEASES
OF THE EAR.

BY WILLIAM MILLIGAN, M.D., A.M.,

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turer upon Diseases of the Ear, the Owens College; Assistant Physician,
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GENTLEMEN,—Purulent inflammation of the mucous membrane lining the cavity of the middle ear may run either an acute or a chronic course. In general the affection begins as an acute process, but in a certain proportion of cases, especially among patients suffering from some form of tubercular disease, the course of events is such that we may fairly speak of the disease as having been chronic from the outset. In such cases the membrana tympani appears as it were to melt away without the presence of pain or of any of the cardinal symptoms of a sthenic inflammation. That purulent disease of the middle ear is a common affection is, of course, known to you all, but although so common, and at times so threatening to the life of the individual, far too little attention is and has been paid to the proper methods of cutting short the disease in its initial stages. A correct knowledge of the etiology and treatment of this affection is also not so widely appreciated as the importance of the disease demands. Suppuration going on in the midst of the temporal bone, in a region surrounded by some of the most important anatomical structures of the human body, is a pathological condition which should command the earnest attention of every one engaged in practising the healing art. That many lives are lost, and much preventable deafness, with all its attendant discomforts and inconveniences, produced either by absolute neglect in the early stages of the disease or by injudicious or inappropriate treatment, is

a fact known too well to every aural surgeon. The actual mortality in this country per annum due to suppurative middle-ear disease is, unfortunately, very difficult to estimate. In the Registrar-General's Annual Reports we find a certain number of deaths attributed to "otitis and otorrhœa," but this number by no means furnishes a correct estimate of the real number of deaths due to diseases of the ear. Numbers die every year from meningeal inflammation, cerebral or cerebellar abscesses, lateral sinus, pyæmia, or general septic processes, whose deaths are classified under these several headings, whereas in reality the primary cause has been some suppurative affection of the middle ear, which has induced one or other of the above-named fatal complications. If we examine the figures relating to the number of deaths returned as due to "otitis and otorrhœa" during the decade from 1878 to 1887, we shall find the following :

Year.	Males.	Females.	Total.
1878	151	116	267
1879	187	106	243
1880	167	115	282
1881	203	156	359
1882	255	198	453
1883	175	168	343
1884	228	168	391
1885	222	198	415
1886	214	195	409
1887	224	184	408

Thus during these ten years 1976 males and 1594 females, or 3570 individuals, perished from otitis and otorrhœa. In the same reports and during the same series of years we find 80,874 deaths attributed to inflammatory affections of the brain and its membranes, and 5336 deaths to septic affections, pyæmia and septicæmia. If we were able in each one of these cases to assign the actual and primary cause of death, the number due in reality to suppurative disease of the middle ear would without doubt be largely increased.

The causes which predispose to purulent inflammation of the middle ear are very various. The following table represents an analysis of the actual cause of the ear affection in 360 cases which have recently come under my care in hospital practice :

	Cases.
Scarlet fever	64
Measles	36
Pharyngeal and naso-pharyngeal catarrh	62
Doubtful (probably catarrhal)	188
Influenza	10
Injury to ear	11
Inflammation of lungs	1
Typhoid fever	3
Typhus fever	1
Tubercular disease of the lungs	16
Teething	11
Bathing in rough water	4
Chicken-pox	2
Whooping-cough	1
Debility following confinement	1
Parotitis	1
Congenital	3

Catarrhal inflammations of the nose, naso-pharynx, and pharynx are probably the most frequent of all causes. Thus, of the above number, if those cases which have been classified as doubtful are taken as being due to such inflammatory conditions (a fair inference, I think, when we consider the exposure to which so many hospital patients are liable), we find 195 out of the 360 cases, or $54\frac{1}{2}$ per cent., due to catarrhal affections of the nose, naso-pharynx, and pharynx. Knapp found 64 per cent. of his cases of middle-ear suppuration due to this same cause. The exanthemata, especially scarlet fever and measles, furnish also a large proportion of the cases. Thus, of the above 360 cases we find 100, or $27\frac{1}{2}$ per cent., attributable to these two causes.

The female sex appears more susceptible to the disease than the male. Of the above 360 cases, 188 were found among females, 172 among males. Others have, however, found the reverse of this, and have stated that the male sex is more frequently affected than the female, in the proportion of three to two. It is possible that the statistics of one clinic may differ somewhat from those of another regarding this point. It is also possible that in one locality the male sex may be more indifferent to the disease than the female, and consequently fail to come under the surgeon's notice.

The right ear appears to be more frequently affected than the left. Of the above 360 cases, the right ear was the seat of disease in 140 cases, the left in 89, and both ears together in 131.

The duration of the trouble varied within very wide limits. The following table is of interest in showing how long the disease had lasted before any advice was sought:

In 86 cases from	1 to 6 months.
" 38 " "	6 months to 1 year.
" 27 " "	1 to 2 years.
" 27 " "	2 " 3 "
" 14 " "	3 " 4 "
" 15 " "	4 " 5 "
" 41 " "	5 " 10 "
" 83 " "	10 " 15 "
" 10 " "	15 " 20 "
" 15 " "	20 " 30 "
" 9 "	over 30 "
" 45 " the duration was doubtful.	

The actual duration of the affection bears an important relation to the prognosis. It is rare for acute cases to be followed by serious intracranial complications. So long as the tympanic mucous membrane remains in a fairly normal condition, so long are its lymphatics and venous radicles able to carry off the putrid products of inflammation to be eliminated by the various emunctory organs of the body. But in those cases which have passed into a chronic condition the state of affairs is totally different. Instead of an intact mucous membrane we find one in which numerous areas of disintegration and ulceration exist. The mucous membrane, having to serve the purpose of a mucoperiosteum, soon transmits the elements of disease to the subjacent bony parietes. Hence we frequently find in such cases denuded and carious areas of bone. The open Haversian canals and canaliculi and the small venous and lymphatic radicles become crowded with micro-organisms, and thus a condition of septic phlebitis and septic lymphangitis is set up. In this way pathogenic organisms find their way into the general circulation and set up at times diffuse, at times localized, septic processes. The part which micro-organisms play in the production of acute purulent inflammation of the middle ear and its complications has been carefully studied during the last few years. Röhrer examined the discharge from the ears of one hundred patients suffering from middle-ear suppuration, and found a great contrast between the organisms contained in fetid and in non-fetid cases. In the former both cocci and bacilli were always found together, in the latter cocci alone were present. In the putrid secretions there were about fifty-eight per cent. of bacilli to forty-two per cent. of cocci, of which one-half were diplococci. In the non-putrid secretions cocci alone were present, of which fifty per cent. were staphylococci, twenty-six per cent. diplococci, nineteen per cent. monococci, and five per cent. streptococci. Baratoux concludes that the streptococcus pyogenes and the staphylo-

coccus pyogenes are the organisms most frequently found in acute otitis, and that all micro-organisms found in the discharges accompanying otitis media are also to be found in the nasal chambers and pharynx. Zaufal has shown that inflammation of the middle ear can result from the invasion of the pneumo-bacillus of Friedländer and the diplococcus of Weichselbaum and Fraenkel. Moos has confirmed these statements, and adds that the streptococcus pyogenes is always found in the most serious cases,—namely, in those attended with mastoiditis and caries.

Netter has also shown that the staphylococcus pyogenes and the streptococcus pyogenes are the prevailing organisms in purulent middle-ear discharges. Kanthack, in an analysis of thirty-one cases of acute otitis media, found (1) in only a few cases pure cultivations of the diplococcus pneumoniae; (2) in most of the cases the diplococcus occurred in conjunction with the staphylococcus; (3) the streptococcus pyogenes was seldom found, and never as a pure cultivation. In chronic cases, also, the diplococcus pneumoniae was never found. From a consideration of these facts we see that in acute otitis media purulenta at least four different micro-organisms play an important part. Whether they are the actual factors in the production of the disease or whether they are merely accompaniments of the suppurative process is a question requiring further elucidation. The part, also, which they play in the production of the various complications, intracranial or otherwise, has not as yet been decided.

The channels by means of which micro-organisms gain entrance into the cavity of the middle ear are as follows:

1. Through the general circulation, as in cases of congenital otitis media purulenta.

2. Through the Eustachian tube, either directly, or indirectly along the nutritive channels of connective tissue which surround the ostium tubæ.

3. Through the membrana tympani, either perforate or imperforate.

4. Through the fissura petro-squamosa along the processes of the dura mater running from the cavity of the skull to the middle ear. These organisms, once having gained admission to the cavity of the middle ear, find in the complicated series of mucous folds and in the seclusion of a cavity more or less shut off from other regions and kept at a fairly uniform temperature a soil highly favorable to their development. Multiplication goes on with extraordinary rapidity, and invasion of neighboring tissues along vascular or connective-tissue channels is prone to take place. In chronic cases, where denuded and carious bone is so liable to be present, the process of invasion takes place with still greater

frequency. The paths along which micro-organisms travel from the middle ear to more distant parts may be classified as follows:

1. Along the small venules and lymphatic radicles which run between the mucous membrane of the middle ear and the meninges of the brain.

2. Along the strands of connective tissue which pass through the roof of the middle ear to the basilar dura.

3. Along the sheaths of the auditory and facial nerves.

In most cases, if not in all, the *materies morbi* which produces both inflammation of the cerebral meninges and intracranial abscess will be found to be organismal. It must be borne in mind that the chronicity of any otorrhœa without the development of any serious complication is no proof that the patient is immune. At any time, either from a chill, an injury, or other such cause, fresh saprophytic poisoning may take place, leading rapidly to disintegration of new areas of tissue, with the result that serious complications may supervene.

Symptomatology.—Acute inflammation of the mucous membrane of the middle ear is ushered in with symptoms which vary in severity according to the intensity of the attack, and the age and the state of general health of the patient. In young children epileptiform convulsions may be induced, and at times a diagnosis of some inflammatory condition of the cerebral meninges is made, a mistake which is frequently not rectified until a stream of pus has appeared from the external meatus, accompanied by the rapid subsidence of all alarming symptoms. This is especially likely to be the case when the attack develops during the course of one of the exanthemata. The general condition of the patient so occupies the attention of the attending physician that the state of the ear is either overlooked or considered as of minor importance.

The most frequent and the most distressing symptom is the presence of pain. Beginning at first merely as a sensation of fulness or discomfort about the ear, it rapidly increases in severity and radiates to the corresponding side of the head. It is referred, as a rule, to the neighborhood of the tragus, and from that point along the course of the Eustachian tube. Every act of deglutition is accompanied by discomfort, so much so as at times to cause the sufferer to forego eating. Sounds also jar upon the ear so that the patient desires the seclusion of a quiet room. In children, where a diagnosis is at times a matter of much difficulty, pressure in front of the tragus frequently furnishes useful information. Pain here may be regarded as almost pathognomonic of middle-ear inflammation.

The temperature at this period varies, but may range from 99° F.

to 102° to 103° F. A slight rigor may at first be noticed, this more especially in young children. The pulsating tinnitus from the highly congested tympanic blood-vessels is intensely disagreeable, and may retard, if it does not prevent, sleep. The amount of deafness present varies a good deal, and depends to some considerable extent upon whether the labyrinth is involved or not. In those cases where this complication has supervened I have found the pain complained of to be more deep-seated and more of a boring nature. Vertigo may or may not be present, and depends probably either upon increased intralabyrinthine pressure, the result of the presence of pent-up secretion in the middle ear, or upon a secondary congestive state of the labyrinthine structures. Other symptoms present are those which ordinarily accompany sthenic inflammation,—*e.g.*, furred tongue, constipation, etc.

Prognosis.—The question of prognosis should be considered from two points of view,—(1) as regards the life of the individual, and (2) as regards the future condition of the ear as an organ of special sense. These two divisions should also be considered according as they relate to acute or to chronic cases. In acute cases, as has already been stated, complications are, as a rule, unusual. In chronic cases, however, especially where the surface of the mucous membrane is disintegrated and ulcerated and where bare bone is present favoring the transmission of organismal infection, the prognosis should be guarded. At any moment infection of new areas may take place, or organisms may be carried to more deeply seated parts, with the result that serious complications may supervene. In acute cases where rational treatment has been adopted it is usual for the ear to regain the power of appreciating and of transmitting sound-waves. In fact, a perfect recovery may, as a rule, be looked for. In rare cases, however, during the course of an acute middle-ear inflammation, secondary involvement of the labyrinthine structures may ensue, resulting in serious injury to the ear as an organ of special sense. The following case illustrates so well this particular point that I venture to lay it before you. H. T., aged forty, caught a severe chill while hunting. The following morning severe pain attacked the left ear, accompanied by constant and distressing tinnitus. A purulent discharge appeared upon the second day, and remained profuse during the following three weeks. On the patient's coming under observation at this time, the hearing power upon the affected side was found to measure $\frac{1}{8}$ of the normal. The tuning-fork placed upon the vertex was heard best upon the affected side, and the duration of bone conduction was good. The left membrana tympani was found perforated in its anterior inferior segment, and a

small granulation protruded through the opening. During the subsequent treatment frequent examinations were made to test the sensibility of the auditory nerve, but, unfortunately, gradual impairment of function was found to be going on. The suppuration was soon arrested and the perforation became cicatrized, but the excitability of the auditory nerve was so reduced that to all practical purposes the ear became a useless organ.

In chronic cases any definite prognosis regarding the future state of the ear as an organ of hearing is a matter of the utmost difficulty and, I may add, uncertainty. Much depends upon the final condition of the ossicular joints and upon the mobility of the foot-plate of the stapes in the foramen ovale, and little if anything upon the situation or size of the perforation.

Objective Appearances.—In the early stages of the inflammatory attack congested blood-vessels are seen running along the handle of the malleus towards the umbo. Later on the radial vessels become engorged, until the whole surface of the membrana tympani assumes a uniform red appearance, in very severe cases becoming almost livid. The light-reflex is soon abolished, and bulging may be seen to have taken place in the posterior segment. In cases where the membrana tympani is peculiarly transparent, formation of pus may be seen to have taken place. Shining through the congested membrane a distinct yellow mass may be observed. In severe cases pulsation of the entire membrana tympani may occasionally be observed.

Pathology.—The pathology of the condition is evident from what has already been said. At first the capillaries of the tympanic mucous membrane are dilated. Gradually, as the intensity of the inflammation advances, they become engorged with blood, so full as at times to rupture and produce the hemorrhagic form of middle-ear disease. Exudation from the engorged blood-vessels, which at first is serous, soon becomes purulent, the pus containing, as has already been remarked, multitudes of micro-organisms. The membrana tympani finally gives way before the gradually increasing pressure of the pent-up secretions, and perforation takes place. The site of the perforation varies, at times being in one segment, at other times in another, most frequently, however, being situated posteriorly.

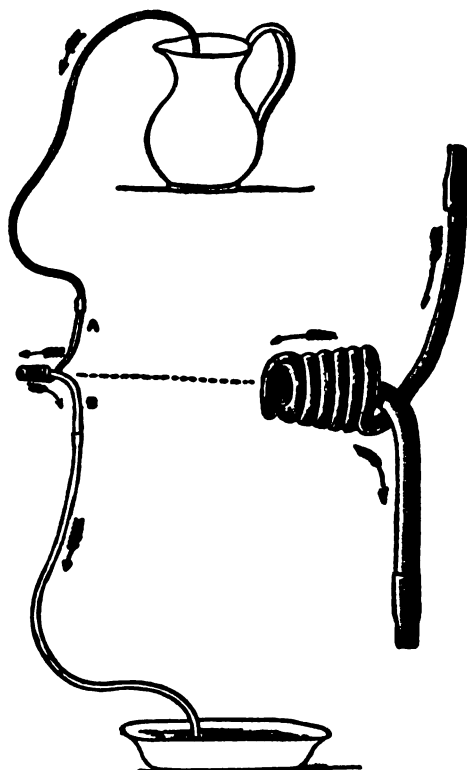
Treatment.—The treatment of acute purulent inflammation of the middle ear should in the first instance be antiphlogistic and at the same time sedative. Our object should be to cut short, if possible, the inflammatory process, or, where pus has already formed, to afford free exit to the discharge. In local phlebotomy we have a means at com-

mand which at times acts like a charm. Two or more leeches applied in front of the tragus, by depleting the part, not only relieve the congestion, but also give very marked relief to the accompanying pain. If it is impossible to secure live leeches, Gorham Bacon's artificial leech may be employed with advantage. The application of heat is of great value. Brau- or sand-bags previously heated may be placed against the ear and changed as frequently as necessary. Poultices, however, should be avoided, as they tend to the production of unhealthy granulation-tissue.

A small irrigation apparatus, as seen in the accompanying illustration, has proved of much value in my hands. A conical spiral made of thin silver tubing, having an in-going tube of small diameter and an out-going tube of somewhat larger diameter,—in order that no excess of pressure may be induced,—is placed in the external meatus and kept *in situ* by a suitably-disposed bandage. A siphon action having been established, a stream of warm fluid circulates through the in-going tube over the surface of the membrana tympani and escapes along the out-going tube. In this way the surface of the membrana tympani is kept continually bathed by a stream of warm fluid, with the result that at times the inflammatory process rapidly subsides.

The judicious use of Politzer's inflating bag at this stage is frequently attended by the most happy results. The closed or partially closed Eustachian tube is opened up, the pressure upon the two surfaces of the membrana tympani is equalized, and in all probability a certain amount

FIG. 1.

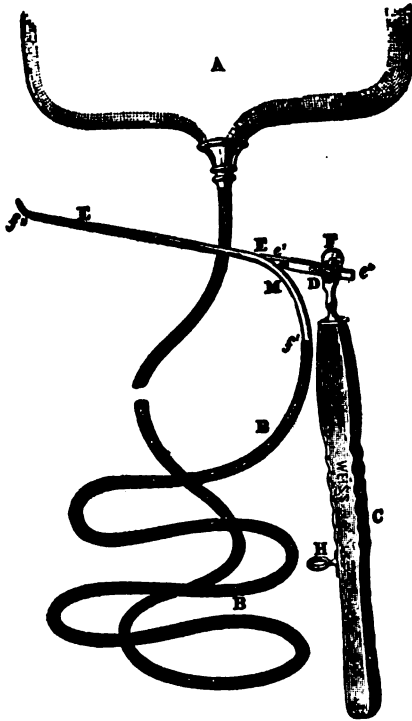


Apparatus for irrigating the middle ear in a state of acute inflammation.

of secretion effects its escape. I would warn you, however, that over-Politzerization may do more harm than good, by driving the germ-laden discharge into the mastoid cells.

In those cases where perforation appears imminent, the surgeon should at once resort to paracentesis of the membrane. Finally, incision not only relieves the patient from much unnecessary suffering, but markedly expedites a cure. The exact site at which a paracentesis should be made varies according to the practice of different surgeons.

FIG. 2.



Intra-tympanic syringe.

If bulging of the membrane is present, the incision should be swept across the most prominent point. Where this is not the case, however, the line of incision may be made either vertically or horizontally. In whatever direction it be made, the great essential is that it should be free. No mere prick is sufficient; a good bold cut is urgently demanded. The old adage, "ubi pus ibi evacua," should be constantly in the surgeon's mind, and the evacuation should be thorough. The employment of Politzer's bag or of Siegle's pneumatic speculum is here indicated in order to drive out or to aspirate the purulent contents from the middle ear. Where the pus is peculiarly thick and tenacious, great advantage will result from a thorough irrigation of the tympanic cavity by

means of a fine intra-tympanic syringe. The accompanying illustration (Fig. 2) depicts such an apparatus, which has afforded me every satisfaction. A is an elevated receiver,—to contain the fluid for irrigation,—which can be raised or lowered according to the amount of pressure desired. E is a fine silver syringe attached to a handle, C, and connected by means of a rubber tube, B, with the receiver A. Under illumination the point *f''* is introduced into the tympanic cavity through the perforation. The fluid is then allowed to circulate

through the system of tubes, and so flushes the cavity of the middle ear of all putrid secretions. If discharge has once established itself, the parts should be kept constantly cleansed. This, of itself, is often quite sufficient to arrest the inflammatory process, but in other cases some antiseptic or astringent drug should be used as well. In acute cases I hold that the less the parts are irritated the better. Hence the employment of dilute acetate of lead lotions, weak watery solutions of resorcin, or dilute solutions of sulphate of atropine, is all that is required.

The *vis medicatrix naturæ* is potent, and simple remedies timely used will answer all the requirements of the case. The ear should be protected by a small pledget of some antiseptic wool, which not only protects the part and absorbs the moisture, but also tends to prevent the entrance of fresh organisms from without. The condition of the pharynx and naso-pharynx must not be forgotten. When there is much hypersecretion the employment of alkaline and detergent sprays is indicated. Rest, warmth, and attention to the condition of the *primæ viæ* are not to be overlooked.

The treatment of chronic suppurative inflammation of the middle ear must depend upon whether we have to deal with an uncomplicated case or with a case in which granulation-tissue, parietal or ossicular caries, cholesteatomatous masses, or secondary affection of the mastoid structures are present. The main requisite here, also, is to keep the parts in as cleanly a state as possible, and to secure free drainage. If the existing perforation appears to be too small for the requirements of the case, it should be enlarged. Any obstacle to the free egress of discharge should be removed,—*e.g.*, granulation-tissue masses, sequestra, etc. Small granulation-tissue masses should be destroyed by the application of some such caustic as chromic acid, nitrate of silver, chloracetic acid, etc. Larger masses should be removed with the snare, or crushed between the blades of a delicate pair of forceps. Carious areas of bone should be scraped or touched with strong mineral acids. To aid in getting rid of all purulent secretion, syringing, Politization, or aspiration with Siegle's speculum should be frequently employed. Recollecting that the purulent secretions teem with micro-organisms, antiseptic solutions should be introduced and allowed to remain in the ear for several minutes at a time, twice or thrice daily. Such drugs as resorcin, sulphocarbolate of zinc or of soda, nitrate of silver, or perchloride of mercury, are in daily use for this purpose. Some surgeons prefer the employment of the dry method of treatment. This consists in the insufflation of such powders as iodoform, euphraphen,

boracic acid, aristol, etc. The ear is first cleansed and then carefully dried, and the powder then insufflated. I would venture to caution you against using powders in (1) acute or subacute cases, (2) in cases where the perforation is small, and (3) in acute cases of perforation of Shrapnell's membrane.

At times it will be found that the suppurative condition of the middle ear is dependent upon the presence of tubercle-bacilli.

Bobone, who has paid special attention to the different forms of otitis media purulenta occurring in tuberculous patients, distinguishes two separate varieties of the disease. In the first the tympanic membrane presents neither thickening nor hyperæmia, but at most a slightly reddish tinge. In the inferior segment there may be a perforation as if made with a punch, its edges smooth and clean, without any tendency to the formation of granulations. In the other form, which occurs among patients suffering from advanced phthisis, the membrana tympani is almost completely destroyed. The mucous membrane of the middle ear is covered by a grayish exudation difficult to dislodge. In old-standing cases there is frequently associated caries of the temporal bone.

Nathan examined the pus from forty cases of otorrhœa. In twelve of these he found the tubercle-bacillus. Other evidences of pulmonary tuberculosis corroborated these results in eight of the twelve cases. Physical examination revealed no pulmonary affection in the ninth case, —that of a scrofulous child. The remaining three were the subjects of caries. Habermann found bacilli in the discharge from four cases of middle-ear disease among twenty-five patients who had died of tuberculosis.

In such cases, in addition to careful local treatment, it is essential that everything should be done to counteract the development of the bacillus. Fresh air, good food, warm clothing, and the internal administration of creosote, guaiacol, or cod-liver oil, are specially indicated.

MOTOR PARALYSES OF THE LARYNX.¹

CLINICAL LECTURE DELIVERED AT THE COLLEGE OF PHYSICIANS AND SURGEONS.

BY GEORGE M. LEFFERTS, M.D.

Professor of Laryngoscopy and Diseases of the Throat.

GENTLEMEN,—I have before me to-day a rather difficult task, for I must condense into one hour what is known at present about motor paralyse of the larynx, and the task is the more difficult as this subject is one which is not to-day an absolutely settled one, and is one about which there are many theories and much needless complexity in your text-books. I hope to simplify this matter for you by throwing aside much discussion and by avoiding disputed points, telling you only what is actually known on the subject.

Let me recall the course of the two nerves which especially concern us to-day,—the recurrent laryngeal nerves. They are, as you know, branches of the pneumogastrics, and receive their motor filaments from the spinal accessory nerve. For the purposes of this lecture I shall regard these recurrent laryngeal nerves as motor nerves; I shall consider that they innervate all the muscles of the larynx except the crico-thyroids and the two little depressors of the epiglottis about which I spoke in my last lecture. We know that these nerves have sensory fibres, but for purely practical and clinical purposes they are motor nerves, and hence we shall consider them as such.

On the left side the recurrent laryngeal nerve is given off from the parent trunk, the pneumogastric, in front of the arch of the aorta, and, winding around this arch, passes back to the groove between the trachea and the œsophagus, ascends the neck, and is distributed to all the muscles on the left side of the larynx except the crico-thyroid and the depressors of the epiglottis. On the right side it pursues an entirely different course, and the practical bearing of this you will hear later. It is given off on the right side from the pneumogastric, passes around the first portion of the subclavian artery, ascends in the sulcus between

¹ Printed from the stenographer's notes.

the trachea and the œsophagus, and enters the larynx, where it is distributed to all the muscles on the right side of the larynx except those already mentioned. The object of this motor innervation is to produce certain physiological movements in the larynx,—abduction, adduction, and tension of the vocal cords. These three physiological movements can be easily carried in your minds. If a stimulus be sent out through the recurrent laryngeals from the brain to the larynx for the vocal cords to be adducted, the arytenoids will be rotated about their delicate joints, and the vocal cords come together in the median line; and similarly, if a stimulus be sent from the brain for the vocal cords to be abducted, the abductor muscles will come into play, the arytenoids will be rotated about their joints, and the vocal cords thrown widely apart, as shown on this manikin. After the vocal cords are adducted and the cartilaginous glottis closed, the tensors may come into play, and by their degrees of tension produce those marvellous changes of pitch and musical note which characterize the human voice. Upon the recognition and recollection of these three physiological movements will depend your understanding of the whole subject of laryngeal paralyses.

If there be a lesion, central or peripheral, sufficient to produce total paralysis of the left recurrent laryngeal nerve, the result will be that every muscle on that side of the larynx, with the exception of the crico-thyroid, will be paralyzed, and the vocal cords will assume what we call “the cadaveric position,” a position midway between extreme adduction and extreme abduction. If the patient attempts to phonate, the left vocal cord will be seen to be immovable, while the right is unusually movable, and passes clear across the median line to meet the paralyzed cord. As the glottis during respiration will be widely opened, there will be no dyspnœa, and there will be no aphonia, because the right vocal cord is able to approximate itself to the left. Where the lesion is so extensive that both laryngeal nerves are pressed upon and their conductivity destroyed, there will be a total paralysis of all the muscles on both sides of the larynx which are supplied by these nerves, and, as a consequence, both cords will be in the cadaveric position; now there will be absolute loss of voice, because the cords cannot be approximated. There will not be dyspnœa, however, for the glottis stands widely open. Recurrent laryngeal paralysis is more commonly observed on the left side, owing to the anatomical relations of the nerve on that side. Aneurism of the arch of the aorta is the most common lesion giving rise to this paralysis, and it has frequently happened that a laryngoscopic examination has given the first clue to the existence of such a condition.

I pass now to the next form of paralysis,—paralysis of the abductors. In considering this subject, I shall be called upon to speak of the respiratory function of the glottis and of a separate ganglionic centre in the brain, which presides over it. Every movement of respiration necessitates a contraction of the posterior crico-arytenoid muscles,—the abductors of the glottis. These muscles are the busiest in the whole body; they are never at rest, and upon their integrity depends the respiratory function of the glottis. It is a curious fact that when pressure is made on a recurrent laryngeal nerve, or upon the parent trunk, at its cerebral centre or in its course, no matter what the nature of the lesion causing the pressure, the motor filaments which run to the abductor muscle are first affected. If both main trunks or both recurrent laryngeal nerves are pressed upon or are diseased, you will early have abductor paralysis on both sides; if only one recurrent laryngeal nerve is affected, there will be paralysis of the abductor on that side only. In either case, through some inherent weakness or proclivity, the abductor filaments are first affected, and abductor paralysis is the result. Later, all the motor filaments become paralyzed, and the vocal cord or cords assume the cadaveric position. As the abductor muscles cannot do their work, the vocal cords will be drawn to the median line by the adductors and will there remain fixed in bilateral abductor paralysis.

What are the symptoms of such a condition? There will be no aphonia, and after a short time there will be scarcely a perceptible change in the voice, because the vocal cords are together in the median line and the adductor and tensor muscles are able to perform their function. How about dyspnoea? The vocal cords are fixed in the median line, and not separated at all during inspiration; hence there is dyspnoea, and this condition is the one dangerous form of laryngeal paralysis. If such a condition occurs suddenly it will produce the most violent inspiratory dyspnoea and necessitate the performance of tracheotomy.

Now I am going to deal with those muscles which adduct the vocal cords. Your text-books constantly speak of paralysis of the adductors, but there is no such thing; there is no recorded case of paralysis of the adductor muscles in the true sense of the term. Every case of adductor paralysis or paresis is dependent upon that peculiar psychical condition which we term hysteria, and is always bilateral. In a case of hysterical aphonia, on attempting to phonate there will be a very slight, irregular movement of the vocal cords; they may be partially adducted, but complete adduction does not occur,

and hence the patient is voiceless. As you know, hysteria never affects the involuntary muscles, and while therefore the voluntary act of adduction fails and the voice is lost, the involuntary acts, such as coughing, are performed as usual. If, then, a patient can cough, but cannot speak, we have a very positive indication of the hysterical nature of the case. You will not fail to note that there is no true paralysis, for there is a decided attempt at adduction. The neurasthenic boy or man, the hysterical girl, or the woman with dysmenorrhœa or ovarian irritation, suddenly loses the voice and as suddenly regains it, and these conditions may exist alternately for a long time. To these hysterical patients with functional aphonia, their ailment is a real one for the time being, and hence it is not enough to tell them that they *can* talk, for they cannot. In speaking of this condition, you should always call it *functional aphonia* rather than use that much-abused term "hysteria."

There is only one other form of paralysis for me to consider on this occasion. We must deal now with the two delicate muscles in the interior of the larynx which we call the internal tensors,—the vocal muscles, the thyro-arytenoid muscles, one on either side of the larynx. By their contraction and relaxation they give to the vocal cords their infinite variations of movement. When these are paralyzed, adduction performed and tension attempted, instead of being tense, the vocal cords are slightly bowed, and on attempting to speak, the current of air rushes through the gap, and either no voice is produced or only a low monotone. This is one of the commonest forms of laryngeal paralysis. These muscles are superficial, lying just underneath the mucous membrane, and are therefore subject to inflammation as well as liable to be overtaxed. This paralysis does not cause complete aphonia, but only the finer gradations of the voice are lost. There is no dyspnœa, because there is no interference with the abduction of the vocal cords.

I have now considered four forms of laryngeal paralysis, and these are the only forms you are likely to meet with clinically. Let me name them once more: (1) total recurrent laryngeal paralysis, whether unilateral or bilateral; (2) that curious form of paralysis which first affects the abductor muscles when the recurrent laryngeal nerve is subjected to pressure, either unilateral or bilateral; (3) adductor paresis, which never occurs except in hysterical patients, and is always bilateral; and (4) paralysis of the tensors of the vocal cords, the commonest of all the four varieties, and one which is usually bilateral.

Now I shall show you a few illustrative cases. This first man's trouble began last May; he has left abductor paralysis. You see there is no dyspnœa and but little interference with his voice. This

next man has left total paralysis, which has existed about one month. His voice is low-pitched and he speaks with difficulty, because only a little over one month ago his left muscles became paralyzed, and his right adductor muscle has not yet learned to do the work of both muscles. After a while his voice will be good. The next man has also left total recurrent paralysis,—that is, his left vocal cord is in the cadaveric position, and the right vocal cord must pass the median line and approximate itself to the left in order to phonate. His trouble has lasted for about ten months, and there is some interference with the vocalization, but not an extinction of the voice. This woman has total right recurrent paralysis,—that is, the right vocal cord is in the cadaveric position, and the left cord is called upon to do the work of both muscles. About three and a half years ago her trouble began, and she first noted that there was difficulty with her voice after reading aloud for some time. You remark that there is nothing very noticeable about her voice, and that there is no dyspnoea. Our last case is a woman with bilateral abductor paralysis; both cords lie in the median line and cannot be separated. Her voice is almost natural, but there has been so much interference with respiration that you see it is necessary for her to wear a tube in her trachea.

[All of these forms of paralysis were also illustrated, as spoken of, by means of a model, upon which tapes to represent the vocal cords were placed in proper position to show the particular direction assumed by the affected cord or cords.]

STENOSIS OF THE LARYNX.

CLINICAL LECTURE DELIVERED AT RUSH MEDICAL COLLEGE

BY E. FLETCHER INGALS, A.M., M.D.,

Professor of Laryngology and Diseases of the Chest, Rush Medical College; Professor of Diseases of the Throat and Chest, Northwestern University Woman's Medical School; Professor of Rhinology and Laryngology, Chicago Polyclinic

GENTLEMEN,—The case I present to-day is one of unusual interest because of its long duration and the fortunate chance which some years ago placed her in the hands of an eminent surgeon, by whom an operation was speedily performed that snatched her from the grave. It is also interesting because of the subsequent history of the case, and the present condition, which has prevented her from speaking for the last five years. She is twenty-two years of age, and I find from the history that five years ago, while suffering from a large goitre, she was greatly troubled with dyspnoea, which at one time became so severe that she was taken by her friends to the hospital, in Milwaukee, in a dying condition. Shortly after entering the hospital she ceased to breathe, but Dr. Senn, who happened to be in the building at the time, was quickly called. By a rapid operation with the thermo-cautery he opened the trachea, then setting up artificial respiration and continuing it for about twenty minutes, he had the satisfaction of seeing her again breathe of her own accord. At that time he removed a wedge-shaped piece of the thyroid. In due time the patient made a good recovery from the operation, but she was still compelled to wear a tracheal tube, and before long it was found that she was unable to breathe through the mouth. She has remained in this condition for the last five years; but recently she came again to Professor Senn, at the Presbyterian Hospital, to see if something could be done to enable her to speak. By him the case was referred to me.

Upon examination of the larynx I find that the vocal cords are free, but there is an obstruction of the trachea a short distance below

the glottis, and she seems unable to get any air through the larynx from the trachea. I find her to-day wearing a trachea-tube such as I have never seen before, but one which has proven very satisfactory in this case, and which I believe would be of very great advantage to some other patients who are obliged to wear a trachea-tube for a long time.

I do not know by whom the tube was invented, but presume that the credit of it should be given to Professor Senn, for it certainly bears the marks of his surgical ingenuity. This device consists of a curved silver tube, about a quarter of an inch in diameter, and two and a half inches in length, inside of a rubber tube of the same calibre, but about five inches long, but which might have been made either longer or shorter, according to the indications of the case. The silver tube maintains the proper curve, and the whole is retained in its position in the trachea without difficulty. To cleanse this tube the patient either removes it or passes a feather down its



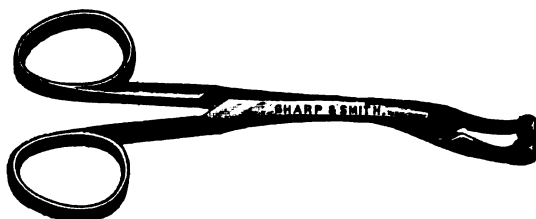
Combined rubber and silver trachea-tube.

whole length while it is in position. I find that the goitre from which she originally suffered has practically disappeared. In removing the tubes and examining the upper portion of the wound I find that the opening between the trachea and the larynx seems to have become completely closed, though we shall probably find a small aperture. We will give her an anæsthetic and endeavor to pass an instrument through the mouth into the trachea, crowding down any tissue that may be obstructing the passage, and then cut it off or destroy it with the galvano-cautery.

Operation.—After the patient had been given an anæsthetic an attempt was made to pass an O'Dwyer tube, and subsequently a pair of forceps from the larynx to the trachea, but the cicatrix was so firm that no safe amount of force would cause the instrument to pass. A forceps was then passed firmly down upon the cicatrix, where it was cut down upon from the external wound, the cicatricial tissue being found about six millimetres in thickness. With a pair of punch-forceps (Fig. 2), devised for this especial purpose, the cicatricial tissue was cut away and the opening enlarged until it corresponded to the size of the

trachea. As there was no instrument at hand to keep the wound open, a piece of rubber tubing, similar in size to that which had been used for the trachea-tube, was split open at the end for about three-quarters of an inch, the sides cut away, and the ends sewed together, so as to leave an opening through which the trachea-tube might be passed. This rubber tube, about an inch and a half in length, was then passed upward from the tracheal wound to the glottis, and the trachea-tube by the aid of dilating forceps was passed through the opening left for it.

FIG. 2.

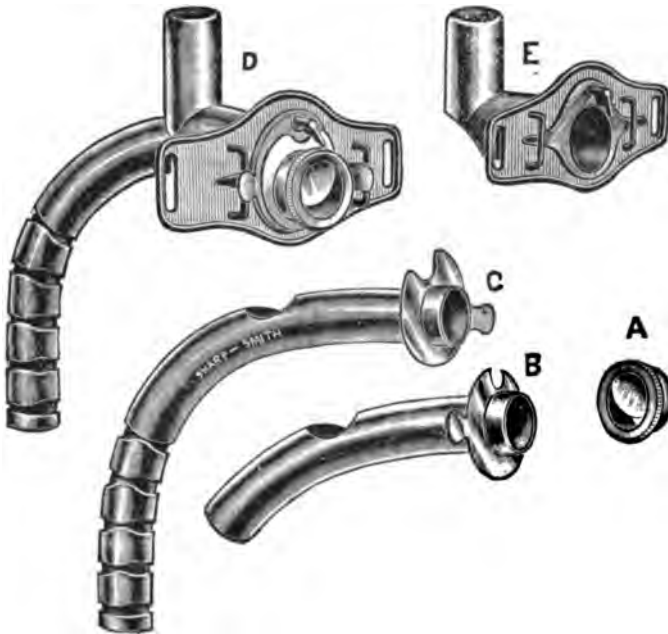


Author's punch-forceps (one-half size).

Subsequent History.—The patient wore this contrivance for a week or ten days while experimental tubes were being made to take its place. Several trials had to be made before the proper angle could be discovered, which proved to be a right angle. A silver tube was then made to pass upward and the combined silver and rubber tube was passed down into the trachea through an opening at its angle. When the wound had partially healed about these tubes, the trachea-tube was withdrawn, and by placing the thumb over the end of the silver tube, which passed upward to the larynx, it was found that the patient, very much to her surprise, could speak distinctly,—the first time for more than five years. It had been found during the course of the treatment that if the trachea-tube was left out for fifteen or twenty minutes it went in with considerable difficulty, owing to contraction of the trachea about two inches below the tracheal opening. Therefore, in order to introduce a permanent silver tube, it became necessary to have one made long enough to reach beyond the elastic portion of the trachea. A tube was devised for this purpose, as shown in Fig. 3. It was necessary, because of its length, to have the lower portion of this jointed like the Durham tube, and it was deemed best that it should pass the curve, from the outer portion of the wound to the trachea, through a middle tube, in order to prevent injury to the tissues when it was introduced after removal for cleansing. While this tube was being constructed I chanced to see in the German exhibit at

the World's Fair a somewhat similar tube with a cap upon the end having a valve to enable the patient to speak. I had a similar cap adjusted to the tube for this patient. This instrument (Fig. 3), for laryngo-tracheal stenosis, consists of an outer tube (E) which passes from the tracheal wound upward to the lower portion of the larynx, a middle tube (B) which passes downward to the trachea, and an inner tube (C) which passes through the middle tube deep into the trachea;

FIG. 3.



Author's laryngo-tracheal stenosis tubes (two-thirds size). A, cap with valve to prevent escape of blast of air during phonation; B, middle tube; C, inner tube; D, instrument complete,—all parts in position; E, outer tube.

also a cap (A) with a valve in it which will allow the air to enter as the patient breathes, and prevent it from escaping, thus allowing her to force air through the larynx in phonation and expiration. The tube which I first had made for this patient was of the same external diameter as the rubber tube which she had been wearing for several years. When it was first introduced she breathed easily, but after about twenty-four hours claimed that it was impossible for her to get sufficient air. The silver tube had a larger calibre than the rubber tube, but there must have been more friction for the air in passing through the lower jointed parts, and it was an eighth of an inch longer than the rubber tube. It was impossible to determine exactly where

the difficulty in respiration lay, and I suspected it was largely hysterical; but she concluded that it was due to the shape of the end of the tube, the rubber tube having been cut off bevelling, and the silver tube having been cut square across. I then had another tube made with a bevelled end corresponding to the shape of the rubber tube. This seemed to the patient to answer the purpose considerably better, but she soon complained again of a dyspnoea and demanded a tube of larger calibre. I then had one made with a calibre nearly two millimetres larger than that of the original tube, and with a bevelled end similar to the one that she had last tried. When this was introduced she found that she could breathe easily and talk without difficulty. She wore the tube for several days with very great satisfaction, but finally gave up the cap at the extremity, as it did not work to her satisfaction, she preferring to place her finger over the end of the tube when she wanted to talk. The patient finally left the hospital and went to her home in Milwaukee. She reported at my office after a couple of months, saying that she could not breathe through the mouth, though she was still able to talk readily when she covered the end of the tube. She regularly removed the inner tube and cleaned it perfectly, and on one or two occasions had all of the tubes taken out by a surgeon and cleansed. At this visit I found the tube going from the tracheal wound up to the glottis nearly closed with tenacious, dried secretions. This was cleaned out with a probe and pledget of cotton, and she found herself again able to breathe easily through the mouth. She was then directed to have a bent applicator which she might pass up into this tube for the purpose of cleansing it daily, and she went to her home satisfied. During the interval since her previous visit to my office the inner jointed tube had become broken, fortunately at a time which did not leave a part of it in the trachea. She was therefore cautioned to be careful to see that the joints were always perfect and the rivets secure. It is probable that she will be obliged to wear the trachea-tube the remainder of her life, because as soon as it is removed contraction takes place about three inches below the glottis to such an extent that respiration is greatly impeded.

RUPTURE OF THE DRUM-HEAD BY BLOWS UPON THE EAR.

CLINICAL LECTURE DELIVERED TO A PRIVATE CLASS.

BY ROBERT BARCLAY, A.M., M.D.,

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LECTURE I.

GENTLEMEN,—I take pleasure in showing you to-day several cases of ruptured drum-head, fairly illustrative of the more striking clinical features of the injury when caused by blows upon the ear. In the lectures upon this subject I shall show you cases where this injury has been inflicted both by the patient, accidentally, upon himself, and by others, in sport and in anger.

The character, as well as the occurrence, of this lesion depends partly upon the organic condition, the trophic changes of the drum-head, partly upon the perviousness of the Eustachian tube, and partly upon the degree of aerial compression produced by the blow upon the auditory canal. Now, since these factors vary indefinitely in the different cases, the character of the lesion will vary, so widely, indeed, as attested by actual observation, that these cases may be said to constitute a panorama of endless novelty; a series of therapeutic problems unique in their refreshing diversity; a field of immense scope for the exercise of judgment and skill.

The injury is often attended by symptoms developed out of all proportion to the lesion and its gravity, and the patient may thereby become

so unduly alarmed as to urgently beg, or, with his relatives and friends, even demand, that "*something should be done to the injured ear.*" I cannot too earnestly admonish you, under such circumstances, to be upon your guard, lest, by injudicious interference upon your part, what might otherwise have proved a comparatively harmless condition should be transformed into one of irreparable damage or even of serious danger to your patient. Pathologically, each case is peculiar, and, from the stand-point of practical therapeutics, unique; so that what is wise in one event is foolish in another, and proper management is not a sport of rule-of-thumb. Obviously a condition of emergency, this injury demands of you prompt and appropriate treatment, which implies, as prerequisite upon your part, thorough clinical experience. Moreover, being often inflicted in an altercation or in anger, it is very apt to prove a bone of litigious contention. The propriety of litigation here will often be determined for the patient or his guardian mainly by the opinions on the case expressed by you, for which you are legally liable and may be held to a strict account; for you will meet these cases frequently, not only in your consultation-room and in the sick-chamber, but also in our courts of justice, both civil and criminal, whither they are wont to resort for vengeance or for the recovery of damages for personal injuries sustained. Here, indeed, while all eyes are turned upon you, your own reputation and the justice of the judgment pronounced by the honorable court are at stake, and will largely depend upon the soundness of the judgment upon which you have based your opinions and utterances. To cultivate sound judgment, then, you will find it necessary not only to study profoundly the lessons of your own clinical experience, but also to carefully compare these with the published opinions and reports of such others as are universally accepted authorities.

To master the relevant facts and to properly prepare yourself for service in this field is a task of no small difficulty. But that you may be encouraged to undertake and pursue it the more willingly and earnestly, I would assure you that there is, perhaps, no other department of medical practice from which you could more reasonably expect or more deservedly obtain reputation and emolument.

Our first patient to-day, Case No. 403,750, is this young man, previously healthy, who, while romping with his fellow-zouaves at the Armory last evening, was playfully, yet not forcibly, slapped upon the left ear by one of them. Momentary vertigo ensued, doubtless from labyrinthine compression or shock; and the hearing of the injured ear has since then been confused by a continuous sound as of escaping steam, and, when he is speaking, by a reverberation of his own voice

in that ear. These latter symptoms are indicative of a disturbance of the equilibrium of tension of the auditory transmitting mechanism, whereby sonorous tissue-undulations, originating at the vocal cords and pharynx, and at the blood-vessels of the ear or its vicinity, and attempting to escape across the bridge of the auditory ossicles and drum-head, meet with abnormal resistance there, and failing, recoil, and disturb the labyrinthine fluid and terminal filaments of the auditory nerve, thus becoming autophonous. There being a variable degree of impassibility of the transmitting mechanism for outgoing tissue-undulations, we might, perhaps, expect to find therewith a like obstruction to incoming ones also, thus impairing the already confused hearing. This indeed proves to be the case in the present instance ; for, as our patient has stated, on entering zouave drill as first sergeant, shortly after the injury, he could not distinguish his captain's fast-manual orders, and was compelled to "come to a 'present' and 'fall out.'"

His deafness, however, was not due solely to labyrinthine shock, confusing autophony, and disturbance of the equilibrium of tension of the auditory transmitting mechanism, as we shall demonstrate later. He complains of numbness of the left ear and its vicinity, doubtless due partly to direct concussion or injury of these parts, and partly to a reflex process in the nerves physiologically connected with them.

He says that while blowing his nose last evening, he "heard the wind whistle through his ear." On Valsalvan inflation now you hear escaping from his left ear a shrill sound, the "perforation-whistle," so-called. Now, this is a physical phenomenon rarely found with traumatic rupture of the drum-head in its early stages, since its perforated wound is usually comparatively large, irregular in outline, and gaping sufficiently to allow escape of tympanic air with but a dull, rushing or blowing sound, if, indeed, not inaudibly to the unassisted ear. With rupture of the drum-head by middle-ear disease, however, the perforation is ordinarily small and circular, so that expulsion of air from the tympanum is usually attended by the characteristic "perforation-whistle." As in the present case, the "perforation-whistle" on Valsalvan inflation, after the reception of a blow by the previously healthy ear, suggests the inference that there is probably a traumatic perforation of the drum-head ; small, linear, and not gaping. Here this is rendered still more probable from the statement of the patient that, during the performance of his ablutions this morning, blood was discovered at the outlet of the affected ear. Let us inspect it. On illuminating the meatus, the first thing abnormal to attract our attention is bright-red blood upon the floor of the canal throughout its entire length, and upon

the lower part of the drum-head, where, beneath the handle of the malleus and lying in its vertical plane, is a dark-red streak. As the patient inflates the tympanum now, by the Valsalvan method, and the air escapes, you readily recognize, in the dark-red streak, a small, linear perforation of the inferior segment of the drum-head, as in this sketch. (Fig. 1.)

With a dossil of absorbent cotton-wool, wet with a sterilized lotion, we will now very gently remove all blood from the visible parts, afterwards drying them thoroughly with absorbent cotton-wool. The indications being to secure coaptation of the lips of the wound, with perfect rest, we will apply a splint to the wound, —a circular piece cut from sized writing-paper, —“the Blake paper disk,” so called after the distinguished inventor of this method, Dr. Clarence J. Blake, of Boston, Massachusetts. While trimming this to a size sufficient to just cover and overlap the little perforation, I would state that Dr. Blake first published this method to the International Otological Society at its first

FIG. 1.



Small, linear, traumatic perforation of the inferior segment of the drum-head.

Congress in New York, in September, 1876. You will find it *in extenso* in the Transactions of that Society, published by D. Appleton & Co., New York, 1877, pages 125 to 132. Our disk is now ready. We take a slender probe, tipped with a small cotton-wool brush which we wet with a sterilized lotion, and then we apply the end of the brush perpendicularly to the centre of the paper disk. The disk adheres to it like the button of a fencing-foil. We now moisten with the sterilized lotion the free surface of the disk, and then carry it very carefully to the perforation, to which we gently apply it, concentrically; pressing it then with a dry cotton-wool brush, or with a silver probe, lightly into snug apposition with the lips of the wound, where we leave it. This is termed the “Blake method” of applying the paper dressing or “Blake paper disk” to a perforation. There are indications and uses for the Blake disk other than for healing perforations (for which it was originally suggested by the inventor), to which I directed the attention of the American Otological Society in 1890. You will find my paper on the subject in their Transactions, published at New Bedford, Massachusetts, in that year. These uses I shall take pleasure in demonstrating to you just as soon as a suitable clinical opportunity presents itself.

The little paper disk which we have just applied will, within a day

or two, become cemented in place by the sizing on the paper and by exudation.

We must earnestly warn the patient against blowing his nose for a few days, lest, by his so doing, air should be forced through the Eustachian tube and through the perforation, and the paper disk be thereby prematurely displaced. If nasal secretions should annoy him, let him rather remove them gently with his handkerchief or else "hawk" and expectorate them; but, I repeat, he must for several days be careful to abstain from blowing his nose. We can leave the disk to be borne onto the canal wall by the out-moving epithelium, or we can remove it earlier with instruments at such time as we shall have reason to believe that the wound has healed. In the present case we may expect a rapid cure, and we shall remove the disk within a few days,—a week at the farthest,—when the perforation will doubtless be firmly closed. After that, at intervals, a gentle Politzer inflation of the tympanum, several times repeated, will doubtless suffice to mobilize the parts, replace the drum-head in its proper plane and curvature, and restore normal intra-tympanic air-pressure.

This patient, Case No. 104,213, is one whom, you remember, we last saw about one month ago, when we requested him to report about this time, so that we might see later the result of the prompt treatment given his severe injury.

It was exactly forty-one days ago that he came to us first. Three hours before doing so he had received, in an altercation, a severe left-hand blow upon his right ear. During the fight which ensued, he was so intent upon punishing his assailant that he was conscious of no aural discomfort or disability. After the fight, however, his "head felt confused," "everything sounded distant," and "there was a hissing sound" in his right ear.

On inspection the right drum-head was found ruptured; almost the entire antero-superior quadrant having been torn away at its radial boundaries and displaced inward, upward, and forward, towards the Eustachian tube. There, folded on its anterior attachment, cord-like at its fold, it lay wholly hidden from view behind its proper segment at the periphery of the drum-head. The visible angular edge of the wound was clean-cut, and there was no considerable blood upon the parts about the perforation, which presented the appearance roughly sketched upon the board. (Fig. 2.) This was indeed a serious injury, and we undertook to repair it at once.

After touching the visible edge of the perforation with a two-per-

cent. aqueous solution of cocaine muriate, and under antiseptic precautions, we replaced the loose flap of the drum-head with a slender, but stiff, rectangular brush of absorbent cotton-wool; and as soon as we had succeeded so that the parts were as in sketch three (Fig. 3), we ap-

FIG. 2.



Traumatic rupture and displacement of almost the entire antero-superior quadrant of the drum-head.

FIG. 3.



Same case as Fig. 2, after replacement of flap of membrana tympani.

plied a paper splint, as in sketch four (Fig. 4), after the manner suggested by Dr. Blake, before described. The patient now began to complain of vertigo and faintness, which disappeared when in dorsal decubitus, returning at once on assuming the sitting posture. Cool and stimulating drinks, with rest supinely, failed to remove this condition; when, within an hour, it was deemed advisable to remove the Blake disk, as by its weight, elastic flexibility, or warping, it might perhaps be exerting undue pressure upon the labyrinth through the intervening ossicles. Notwithstanding this procedure, however, and the subsequent occasional administration of mild stimulants, the vertiginous and syn-copal symptoms invariably recurred on resumption of the sitting posture. This condition persisting for several hours more, a consultation was held upon the case, with Dr. F. V. L. Brokaw, with a view to ascertaining the exact extra-aural cause of this disturbance. A very careful and thorough physical examination was made. The attempt to extract definite and unrestrained statements from the patient as to his previous condition and habits was more successful than before. It was finally thought that the condition was essentially one of general nervous exhaustion from causes other than the aural injury, and the patient was soon relieved with aromatic spirit of ammonia and hypodermic injection of strychnine sulphate. On the following day the perforation appeared as in sketch five (Fig. 5). At that time it was found that he could hear a forty-eight-inch watch at a distance of twelve inches with the left ear, eight inches with the injured ear.

Now, in order to estimate the hearing-power, we first measure the

distance from the ear to the source of a sound of fixed intensity, when just barely within range of certain hearing, having by numerous tests previously ascertained the average hearing-distance for the healthy ear with this same sound. We now compute the square of each of these. Inasmuch as the intensity of sound varies inversely as the

FIG. 4.



Same case as Fig. 3, after application of a paper disk as splint.

FIG. 5.



Same case as Fig. 4, on the day after receipt of the injury and replacement of the flap of membrane.

square of the distance from its source, and hearing varies directly as the intensity of the sound, we find the hearing-power of an ear equal to a fraction whose numerator is the square of the actual hearing-distance of this ear for the testing-sound selected, and whose denominator is the square of the average hearing-distance of the normal ear for this same sound. Thus, here we have for the hearing-power of the left ear (if determined with a watch heard by the normal ear at an average distance of forty-eight inches), $(\frac{1}{4} \frac{1}{2} \text{ or } \frac{1}{4})^2 = \frac{1}{16}$: one-sixteenth of normal hearing-power; while of the injured ear $(\frac{2}{8} \text{ or } \frac{1}{4})^2 = \frac{1}{8}$: one-thirty-sixth of normal hearing-power; certainly marked deafness for the watch.

The high c'' tuning-fork was heard by air-conduction longer with the right than with the left ear, the converse being the case when the test was made with the middle c' tuning-fork,—an acoustical paradox, still further interesting when compared with his hearing for Koenig's rods. From these he heard forty thousand vibrations a second with the left ear; with the injured ear, forty-five thousand. Now, it is known that the range of hearing for very high-pitched tones is often extended by perforation of the drum-head of a healthy ear; that tones still more highly pitched can then be heard; from which some authorities have made the curious inference that the transmitting mechanism of the normal ear is actually an obstacle to certain sound-waves which the inner ear could otherwise enable us to hear. Why this is so is a question whose answer may perhaps not seem so difficult to deter-

mine if we first take into consideration the physiological fact that from childhood normal hearing at any given time is of higher range among the upper tones than at any subsequent period of life.

Our patient, fearing a recurrence of his vertiginous and syncopal symptoms, would not allow us to reapply a paper dressing to the perforation; so a very light cloud of powdered boracic acid was insufflated upon the parts.

Upon the third and fourth days after the injury, the edge of the wound was barely touched with a cotton-wool dossil moistened with an aqueous solution of silver nitrate, twenty grains to the ounce. The perforation upon the following day appeared as in sketch six (Fig. 6).

Upon the sixth day after the injury, he could hear the forty-eight-inch watch with the injured ear, at sixteen inches' distance; twice as far as, and showing fourfold better hearing than, upon the day after the

FIG. 6.



Same case as Fig. 5, on the sixth day after receipt of injury, or fifth day after Fig. 5.

FIG. 7.



Same case as Fig. 6, at time of application of second Blake paper disk.

injury, when he heard it at eight inches. The perforation was now as in sketch seven (Fig. 7), when the patient was prevailed upon to permit the application of the Blake disk, which was accordingly effected at once.

The disk being properly in place upon the seventh, it was allowed to remain until the eleventh day, when it was thought proper to remove it; after which we applied aqueous solution of silver nitrate, twenty grains to the ounce.

Finding the perforation firmly healed on the next or twelfth day, the tympanum was gently inflated by the Valsalvan method.

On the thirteenth day we dismissed the patient, requesting him to report again,—as he has done,—within one month.

His hearing is normal; and on inspecting his ear to-day, which is just forty-one days since his injury, you see that no trace whatever of the lesion is recognizable; thus attesting conclusively the wisdom of giving prompt and appropriate treatment to a traumatic rupture of the drum-head.

This patient, Case No. 404,355, is fifteen years of age, and came to us, you remember, about three weeks ago, stating that his right ear had been injured by a blow inflicted on the preceding day. While crossing a vacant lot, at that time, to elude some larger boys who were tormenting him, he was waylaid, as he passed, by one of them, who dealt him a fist-blow directly upon the right ear so forcibly as to fell him. Pain in this ear ensued, with numbness, tinnitus, and autophony; and hearing was so far impaired on that side that when at three feet distance he could only hear with the injured ear a loud voice, so loud, indeed, that he might perhaps have heard it with the uninjured ear, instead, although tightly stopped with the finger. He bathed the injured ear with cold water, although no aural discharge had been recognized. At his first visit to us he could with his left ear recognize the tick of a forty-eight-inch watch at a distance of several feet, while with the injured ear he could hear it only when pressed tightly against it. In the injured ear, the middle *c'* tuning-fork was heard longer by bone-conduction than by air-conduction; and with this fork, hearing by air-conduction was not as good in the injured as in the uninjured ear. But with the high *c''* tuning-fork, the hearing by air-conduction was the same in both ears. Moreover, on testing with Koenig's rods, it was found that, although forty thousand vibrations a second were only uncertainly and indistinctly heard with the uninjured ear, yet with the injured ear they were readily heard with certainty and distinctness. Thus, while the hearing of the injured ear for low-pitched tones had been impaired, that for high-pitched ones was at least as good as, even if it were not indeed better than, it had been before the injury. Hence, for reasons explained with the preceding case, we naturally expected to find a perforated drum-head. And in this expectation we were not disappointed; for, on inspection, we discovered a very large, heart-shaped perforation, concentric, so to speak, with the drum-head. The mucous membrane of the promontory seemed normal, save for the presence of a dilated blood-vessel leading from the promontory towards the Eustachian tube. Of the drum-head only a narrow rim remained *in situ*, dotted with bright-red spots very like blood. The anterior edge of the perforation was somewhat regularly circular, while the posterior was quite irregularly so; and the latter joined the edge that descended behind the malleus-handle at an acute angle, not in a curve, as it would have done if the perforation had been an old one. The short process of the malleus marked but a light-yellow spot at the flaccid membrane, which was throughout of a uniform cardinal-red color. This intense redness extended downward to the extremity of that portion of the drum-head

attached to the handle of the malleus. This remnant membrane had swung, or folded, inward, towards the inner tympanic wall, the posterior flap only being visible, thus rendering the mass club-shaped. The parts appeared as in sketch eight (Fig. 8). Owing to swelling and ad-

hesion of the displaced parts, all attempts to replace them proved fruitless; and, as such repeated endeavors served but to cause the patient cold-sweating, thirst, nausea, and faintness, they were discontinued, and the canal was left stopped with a wad of absorbent cotton-wool.

FIG. 8.



Traumatic rupture of large portion of drum-head, the displaced portion wrapped inward, forming club-shaped mass on inner aspect of malleus-handle.

The other ear and the naso-oro-pharynx presented no appearance pathologically remarkable. The patient, being accustomed to daily "cycling," was, in prudence, requested to keep off his "wheel" until permitted to do otherwise.

On the second day after the injury he could, with the wounded ear, hear the forty-eight-inch watch on contact without pressure. His mother, who accompanied him, informed us that he had never suffered from ear-disease but once, and then, when about four years of age, with ear-ache only, and that but for a day; and states that she had never known of his having had any aural discharge whatever at any time.

Two days later he could, with the injured ear, hear a slightly elevated voice at twenty-five feet distance, and a loud whisper at thirteen feet. The ear seemed less abnormal, the drum-head having cleared somewhat, the blood-spots grown much darker, and the outline of the perforation become smoother and more circular.

On the fifth day after the injury the parts had still further improved; so we directed the patient to keep the ear stopped with absorbent cotton-wool, to avoid undue exposure and active exercise, and to report to us at short intervals; for we hoped to find some further evidence of an attempt at reformation by the drum-head. Touching the edges of the perforation with escharotic solutions, and various other methods of attempting to stimulate the growth of membrane in the perforation, have been carefully tried and have proved ineffectual. Inasmuch as the edge of the heart-shaped perforation at the lower end of the handle of the malleus lies in a deeper plane than that of the greater part of the outer edge which encloses it, the Blake disk has been unavailable for our purpose. We might, perhaps, try, later, a "Yearsley-pellet" or cotton-wool wad, worn continuously against the lower end of the malleus and edge of the perforation. To this and

various other stimulants of growth, we shall give a fair trial and report results to you. But the prospect of success seems at best so very unpromising that I think the lesion of the drum-head in this case may safely be pronounced a permanent disability, and the case incurable; and if it prove so, it will be mainly because of the unfortunate neglect to give prompt attention to the ear immediately after the receipt of the injury. An instructive case, this, when its distressing results are compared with the perfect cure which crowned our prompt efforts in the preceding case.

Dermatology.

PLICA POLONICA.

CLINICAL LECTURE DELIVERED TO THE SENIOR CLASS OF THE MARION-SIMS
COLLEGE OF MEDICINE.

BY A. H. OHMANN-DUMESNIL, M.D.,

Professor of Dermatology and Syphilography in the Marion-Sims College of Medicine, St. Louis.

GENTLEMEN,—I purpose speaking to you to-day on a condition which is comparatively rare in this country, although common enough in some parts of Europe. It is a peculiar disease which has been universally attributed to filth and want of proper care. Every author who has written upon this subject, with the possible exception of one or two, has agreed in this view of the etiology of the trouble. This, no doubt, has been due to the fact that nearly all the cases observed, described, and figured had the same characteristics in common,—viz., a matting of the hair in an inextricable mass, accompanied by a greater or less inflamed condition of the scalp, exuding a glairy fluid, possessing a more or less pronounced foul smell, the whole accompanied by the presence of a large number of animal parasites. The general want of cleanliness in those in whom it has generally been observed has tended still further to confirm this idea. The trouble has been observed most often in Polish women, whence it derived its name of *plica polonica*. The matting of the hair does not necessarily implicate the entire scalp, but may be confined to even a single lock of hair of greater or less dimensions, forming a hard, cylindrical, filthy mass, so characteristically denominated as *zopf* by the Germans.

It may not be known to you that such specimens are almost impossible to procure on account of the prevalent superstition entertained by those affected in this way. They are firmly convinced of two things: first, that if the matted hair be cut off blood will ooze out at the site of incision; second, that the ultimate result of such removal will be death. This is an old belief which still remains in full force up to the present day, and I have had occasion to note it in cases occurring in this city.



FIG. 2.—External aspect of hair-mass.



FIG. 3.—Internal aspect of hair-mass.

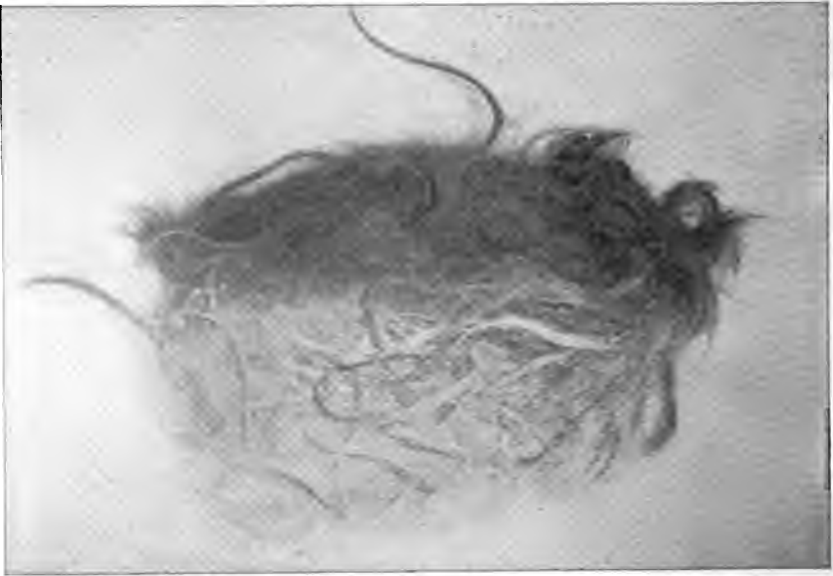


FIG. 4.—Upper or outer surface of mass of hair.



FIG. 5.—Lower or inner surface of mass of hair.

But, to recur to the case in hand, which is the second of its kind which I have had the good fortune to encounter. It is a typical case minus some features noted in the filthy cases, but it is of interest as throwing some light upon a point to which but one other investigator besides myself has drawn attention. I am indebted for the case and its history to Dr. M. B. Croll, of West Point, Nebraska. The history of the case may be briefly summarized as follows :

T. M. is a girl of twelve, of German extraction, slight, puny, and sickly. She is very "nervous." She has suffered from most of the diseases of childhood, such as measles, whooping-cough, varicella, pneumonia, and, later on, of chorea in a mild form. She has never menstruated. She became ill, having contracted remittent fever, of a bilious form, which followed the usual course, and she was convalescing at the end of the third week of her illness. She was treated by the family physician about ten days, when he left on a trip to the World's Fair, leaving Dr. Croll in charge. When the announcement was made to the patient that the family physician was going away she became very nervous, and that night became more delirious. On the next morning, when Dr. Croll first saw her, her temperature was higher, registering 102.6° F. She was decidedly nervous at meeting the "new" doctor. In the evening her temperature was 104.8° F. She had been very restless during the day, being slightly delirious at times.

On the third day of Dr. Croll's visits to the patient her hair on the back of the head began to mat to such a degree that a comb could not be used. This condition persisting the hair was cut off ten days after it had begun to felt. There is not much more to add. After the patient left her bed her health improved and she has been better than for years past. Her hair is falling out rather profusely, this being the old growth which is rapidly supplanted by a new and more vigorous one. Of course, there will be no alopecia.

It may not be uninteresting to add that the patient's skin has always been apparently normal, there being no seborrhœa, eczema, or other cutaneous trouble.

I have here a mass of hair removed from the posterior portion of the patient's scalp, including the two braids. Upon examining this you will find that its external or posterior aspect shows that the hairs are tangled up in an apparently inextricable mass. Upon its inner aspect the condition is still more marked. Here there is a veritable felting of the hair with a small curl near the centre. This is the condition which has given the condition the name of *plica*, from *πλικο*, "I fold." In the specimen before you there is no evidence of filth, dirt, or

parasites, and it resembles greatly this other specimen which I have described on a former occasion in the *International Medical Magazine* for July, 1893. Both are derived from girls and both are blondes. In the present case the plaits are unaffected. They do not present any matting of the hair or felting, except within a very short distance of their origin from the scalp.

I have not the time to enter into a detailed statement of the complicated classification made by some authors respecting the varieties of this trouble. Those who are curious on this point may consult Ali-

FIG. 1.



Hair from mass cut off.



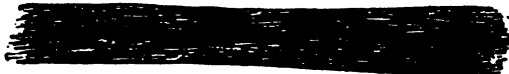
Hair one month after attack.



Hair two months after attack.



Hair three months after attack.



Hair five months after attack.



New growth five months after attack.

Microscopic appearances of hairs.

bert and other ancient authors of works on dermatology. The point of greatest interest is that concerning the etiology of this affection of the hair. As I have already stated, filth is most generally regarded as the underlying cause. Again, inflammatory or phlegmonous affections of the scalp have been advanced as possible causes, as well as various animal and vegetable parasites. The present specimens, however, place all these alleged causes beyond probability.

An investigation pursued in a former specimen led me to make the same examinations in the present, and with analogous results leading

to the same conclusion. If we take hairs derived from the mass it will be found that changes in the hairs themselves have taken place. As you may see by microscopic inspection the changes affect the medulla of the hair. You will note that in some specimens (the older ones) there are considerable losses in this structure, the most marked being when the process is at its height. As the condition improves these losses become less and less, until we once more find a normal hair somewhat smaller in calibre on account of its recent growth. This condition I also observed in my former case; but there was in addition a bursting of the hair shaft in some instances, this being a veritable trichorrexia. In the present instance this has not been observed, although a persistent search might possibly reveal it.

The question which naturally arises is as to what this indicates. It certainly shows a disturbance of nutrition, and such disturbance of nutrition in the pilous system is indicative of tropho-neurotic disturbance. I am not alone in this opinion, for it is shared by the only other author who has ever investigated this subject thoroughly,—S. Jarochevski. This neurotic origin is confirmed by the history and condition of the patient. We find that she was of a nervous disposition, and she suddenly developed an intensified attack so marked that delirium supervened. The marked nervous explosion was followed by the trichoma, which seemed to increase for some little time. The condition of lack of nutrition, however, diminished *pari passu* with returning health, until now, as you may observe, the hair is normal, both upon inspection and microscopic examination.

A question which will naturally arise is as to the proper management of such cases. So far as the local treatment of such cases is concerned, removal of the entire mass of matted hair is absolutely essential. It cannot be untangled; it may prove a nidus for dirt and parasites, and it is a mass of perfectly useless hair. Having closely cropped the scalp a local stimulating application will tend to increase the circulation of the scalp and, in this way, the nutrition of the hair. A good application would be one after the following formula:

R Resorcini, ʒi;
Hydrarg. bichloridi, gr. ii;
Alcoholis, ʒvi. M.
Sig.—Apply twice a day.

This, however, is not sufficient. The *fons et origo* of the trouble must be attacked. The nervous system must be brought up to par. You will find that this involves a problem which is not devoid of com-

plications. No hard-and-fast rules can be laid down which will cover all cases. Neurotic disturbances are numerous and varied, and demand, each one in turn, appropriate treatment, which can only be determined by the symptoms presented by the case in hand. Nerve-tonics, stimulants, sedatives, alteratives, and other means are indicated in different cases. The removal or abolition of central or peripheral exciting causes of nerve-disturbance may be necessary. General measures of various natures are frequently called for, and it is not unusual to have the necessity shown of remedying certain diseased states before the nerves will regain their normal tone. To enter into but a superficial consideration of these topics would occupy too much time. It must not be forgotten, however, that the general means indicated should be followed in order to obtain satisfactory results of a permanent nature. The patient before you is a living proof of the efficacy of such measures obtained within a comparatively short period of time.

In conclusion, do not forget that investigation turned into the proper channels will yield a rich harvest, and will lead to useful and valuable deductions, not only in the field of pathology but in that of etiology as well. And etiology is the key to rational therapeutics which must inevitably lead to successful results.



A case of lupus vulgaris.

LUPUS VULGARIS.

CLINICAL LECTURE DELIVERED AT THE POST-GRADUATE SCHOOL.

BY WILLIAM L. BAUM, M.D.,

**Professor of Skin and Venereal Diseases, Post-Graduate Medical School of Chicago;
Fellow of the Chicago Academy of Medicine.**

GENTLEMEN,—The patient whom I am about to show you to-day is a woman, thirty-six years of age, a Norwegian by birth, who first appeared at this clinic in September, 1892. She presented the following characteristics at that time: An ulceration, quite sharply defined, reaching back from the external canthus of the eye on each side to the base of the ear and running down in a serpiginous manner on either side, following the line of the clavicle to the sternum. There was ectropium of the lower lids of both eyes. The open ulceration extended to a line of about one-half inch below the nasal opening and almost completely encircled the mouth, without any involvement of the mucous membrane. There was total destruction of the cutaneous surface and the cartilaginous portion of the nose. The patient gave the following history.

About twenty-one years ago, while living in a small village in Norway, her mother noticed a small reddish patch near the ala of the nose, on the right side of the face. This did not attract any attention, but, as it was found that it not only did not disappear, but that, on the contrary, it increased very slowly, she was taken to a local physician about two years after the first involvement. The patch was treated at that time evidently with an arsenical paste, and seemingly the patient made a good recovery. About six months thereafter she noticed on the left ala of the nose the same characteristic disturbance taking place. This increased with rather more rapidity than the first named, and upon either cheek there appeared isolated, small, pin-head nodules. About eighteen years ago the patient was taken by her parents to Professor Boeck, at Christiania, for treatment, and he pronounced it a case of lupus vulgaris. At that time the process had extended so that it completely covered the cutaneous surface of the nose and involved

also the cartilaginous portions of the nose, presenting a tuberculated appearance. An operation was advised, consented to by the parents, and the process entirely removed with the knife, the surface afterwards cauterized, and the patient had again a period of several years of comparative immunity from the disease. About fourteen years ago the process recommenced, and has been spreading ever since that time until it has reached its present dimensions. At the time she came to the hospital the base of the ulceration was reddish in color, bled easily, and excreted a serous exudate with an admixture of pus. The edges were covered with hard, dry incrustations, rising a quarter of an inch above the surface of the surrounding skin. Beyond the margin of this ulcerative process were found a number of small nodules, situated below the epidermal covering, that is, in the corium of the skin. They were about the size of small grains of shot, and gave somewhat the sensation upon touch that would be produced by a small, hard body felt through a membrane.

Under the name of *lupus vulgaris* is understood a disease which is characterized by the appearance of small nodules, deeply embedded in the corium of the skin, which have a tendency to undergo exulceration, leaving a peculiar cicatricial tissue behind. These nodules, which are characteristic of lupus, having existed for some weeks or months, undergo some characteristic changes. Sometimes they undergo a fatty metamorphosis, the surface sinks in, the epidermis becomes wrinkled and scales off. This condition is known as *lupus exfoliativus*. Again, after resorption there sometimes is left a flat depression with cicatrices, or with this exfoliative process a superficial ulceration ensues, which is called *lupus exulcerans*. This usually occurs in the larger patches, and where there is considerable destruction of tissue we have *lupus exedens*, such as you see in the nose of the patient before you. Sometimes there are around the edges of these ulcerations, on account of the drying up of the discharge, large incrustations, and sometimes even excrescences on this reddish bleeding surface, which, upon drying into hard wart-like incrustations, are known as *lupus papillaris verrucosus*; these usually occur upon the extremities. Again, there may be, after a number of recurrent attacks, a sort of elephantiasic thickening and hardening of the skin, what is called by the French *lupus sclerosis*.

There always has been and still exists a considerable divergence of opinion as regards the etiology of this disease. Some authors take the ground that either syphilis, scrofulosis, or tuberculosis is the causative element in the production of this disease. It has been thought at times

that it is a form of hereditary syphilis. We often find patients suffering from lupus after contracting syphilis, and the syphilis running its course irrespective of the lupoid condition. This was quite a natural error if we take into consideration the serpiginous character of the ulcerations; but it is known that syphilis manifests itself as syphilis always and by unmistakable characteristic symptoms, whether it be from a primary infection or as a result of the hereditary transmission.

In regard to its relationship to scrofulosa it would hardly be necessary to go into further details, as, under such conditions, we would expect to find the patient had a scrofulous cachexia; but lupus is seldom found in such individuals. The most important of these supposed causative elements is the tubercle bacillus. Many authors have for a long time seemingly demonstrated the direct relationship between lupus and tuberculosis. At the time of the discovery of the giant cells it was almost universally conceded that, as the lupus nodule also contained giant cells, it must necessarily be a sort of modified local tuberculosis. But, as Virchow pointed out, giant cells are to be found in most of the granulomata, in the induration of hard chancres, in sarcomatous processes, in normal granulations, etc. But it was not until the discovery of the tubercle bacillus by Koch, and the demonstration by Demme of the presence of this bacillus in lupoid conditions, that it was generally acknowledged as a tubercular process. Unfortunately for the advocates of this theory, tubercle bacilli have been found in only a small proportion of cases of lupus and usually in the giant cells, and rarely there in any quantity. This is of some importance in view of the fact that similar bacilli have been found in syphilis and in cases of leprosy. I have made a number of microscopical sections of the nodules from this patient and from one other patient in our clinic, and have never yet been able to demonstrate the presence of the tubercle bacillus in a lupoid nodule which had not undergone the process of retrograde metamorphosis; although Furth has found a growth of eight tubercle bacilli in one cell of the rete in a specimen taken from a patient in Kaposi's clinic.

If we take into consideration that the vast majority of these cases of lupus occur in individuals who are otherwise in the best of health, and the fact that but few of them suffer from general tuberculosis or other tubercular conditions, I think it would be manifestly improper to class lupus as a tubercular condition, for there is a true tuberculosis of the skin with characteristic symptoms, running a course which hardly resembles in any of its details the characteristic lupoid process.

In regard to the prognosis, this condition is not a serious one in so

far as its danger to life is concerned, but it may exist for years, and especially in those cases which occur in children of from six to thirteen years, and are characterized by successive recurrences, the prognosis as to the future is not good. It is manifestly the duty of the physician to eradicate the trouble as rapidly as possible before extensive destructive changes have taken place, such as are seen in the patient before us. Here is a woman, otherwise a normal human being in every detail, in the best of health, who, on account of the mutilation caused by this disease, is practically ostracized from association with other human beings. She never appears without some covering over her face.

It may be well to outline what would constitute a practical method of treatment for these cases before they have reached the alarming proportions that this one has. In cases of lupus disseminatus, where the individual nodules are scattered about over the surface of the skin, they can be destroyed either by touching them with a Paquelin cautery, or preferably with a sharp solid stick of caustic. It will be found that the point of the caustic will sink into the corium of the skin through the lupoid nodule just as the point of a pencil penetrates a piece of wet card-board. This will leave a small cicatrix which is not so disfiguring to the patient as the process would be if allowed to remain. For larger patches of lupus discretus I would advise scraping of the base and edges with a Volkmann spoon and the application immediately thereafter of an ointment of twenty per cent. of pyrogallie acid (pyrogallie acid twenty parts, vaseline and lanolin forty each), this to be left applied for forty-eight hours, and the wound afterward dressed with either a five-per-cent. iodoform ointment, or preferably a ten-per-cent. aristol. This dressing must be removed every second day, and the edges scraped down to the level of the surrounding epidermis with a Volkmann spoon, the granulations in the centre of the wound being destroyed by a ten-per-cent. solution of nitrate of silver, this process being repeated, so that the process of healing and the repair of the epidermal surfaces should take place from the periphery to the centre of the wound. If this method of treatment is carefully carried out we will find that the scar which is left has a smooth appearance; there are not the usual layers and ridges of cicatricial tissue in which we expect to find the characteristic lupoid nodules recurring. The arsenical paste can be used, and after the destruction of the lupoid spot the treatment above outlined can be carried on to good advantage.

A number of cases have been treated by subcutaneous injections around the lupoid spots with chloride of zinc, first anæsthetizing the part with injections of cocaine. This, however, while producing ex-

tensive destruction of the nodules and surrounding skin, leaves bad cicatrices, and is objected to most forcibly by the patient on account of its painful character.

The method of treatment which we have employed in this case was somewhat of a variation from the ordinary methods of treatment. In a case where such a large territory has been involved it is impossible to totally extirpate the disease, and the physician can consider himself fortunate if he is able to arrest the progress of the disease and prevent its spread to new parts; in other words, to keep it in a state of *status quo*. That is what we have attempted to accomplish in the case of this patient. As she had been treated by almost every known method of treatment, and at the hands of able dermatologists, and as she has objected to any treatment whereby she would be compelled to remain in a hospital, we applied first a ten-per-cent. ointment of ichthyol, more particularly for its stimulating effect on the large ulcerated patches. This was followed by the use of a ten-per-cent. aristol ointment. There was some tendency towards repair, but the progress of the case was very slow. About four months ago we applied an ointment composed of the following: Alumol, twenty parts; lanolin, ten parts; vaseline, seventy parts. The alumol ointment was given on account of its antiseptic and astringent action, and the results were more gratifying than those previously obtained by the other methods of treatment.

In this photograph—which was taken two months ago—you will observe that the ulceration has healed up, leaving a comparatively smooth surface, and some incrustation at the margins of the old ulcerations, which has rapidly disappeared under this treatment. We at one time made applications of alumol in fifty-per-cent. solutions, but it was not as efficacious as the alumol in the form of an ointment, and necessitated the patient's remaining in doors for the time being. The patient has received no internal treatment, and, in fact, such treatment is not necessary unless the general condition of the patient demands it.

We have not used Koch's injections of tuberculin on this patient, in view of the fact that its seemingly curative action has not been followed by any permanent benefit to patients, inasmuch as most of those dermatologists who have used it on patients suffering from lupus report their cases as having relapsed within a certain length of time after the method had been employed. I believe that the action of Koch's lymph is simply analogous to that which is exercised by the toxic agent generated by erysipelas when erysipelas occurs during the progress of some other cutaneous affections, and it is known that even some malignant growths have disappeared when attacked by erysipelas.

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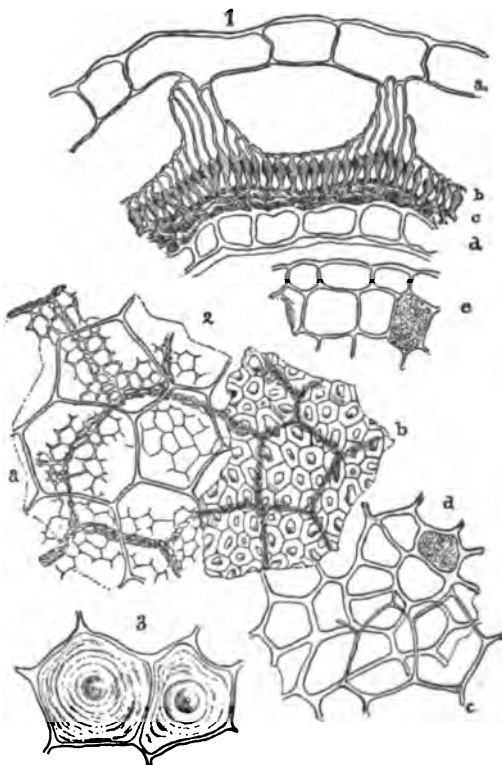
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1. Transverse section of the shell of a seed: a, epidermal layer with cells empty; b, connecting layer; c, layer of pigment-cells; d, parenchymatous cells; e, embryo. 2. Fragment of seed from a surface point of view; parts as in 1. 3. Cells from the epidermal layer, full of mucilage.

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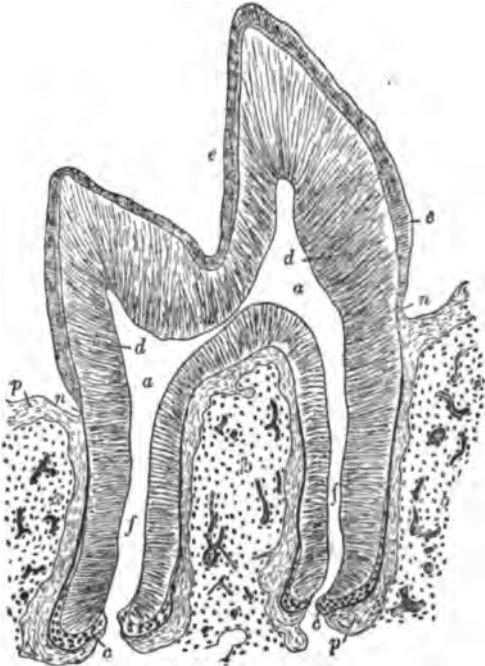
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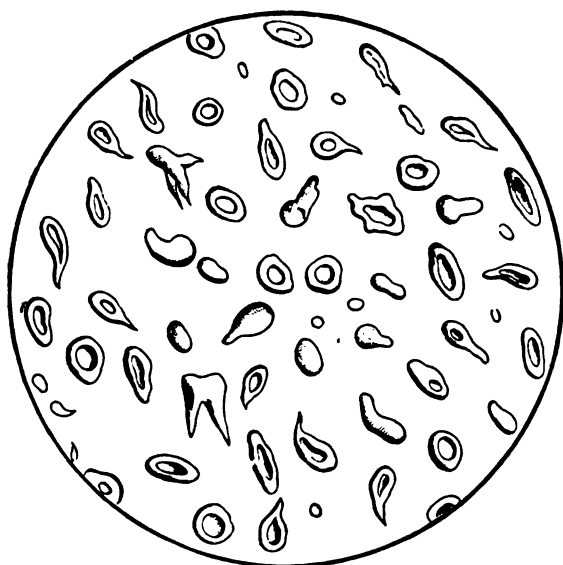
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[Illustration from chapter on "Diseases of the Blood."]

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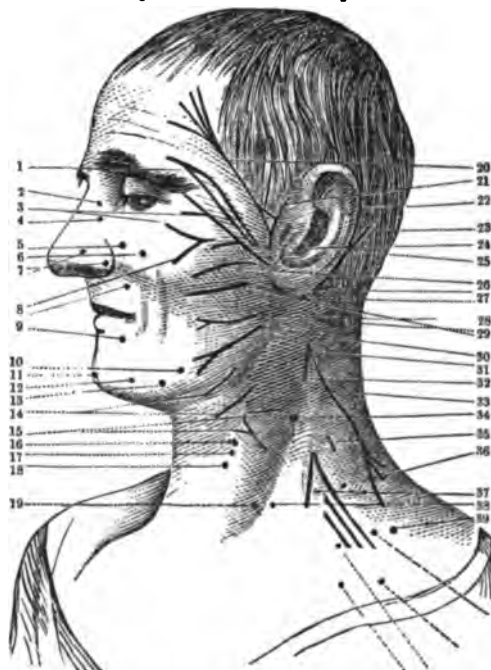
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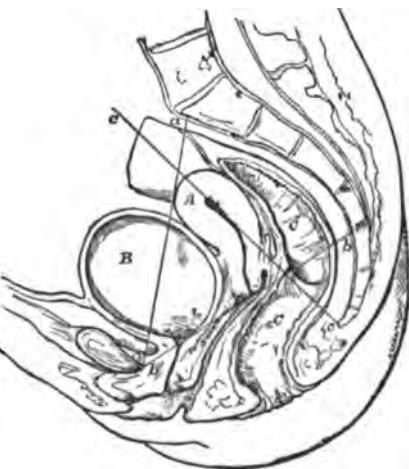
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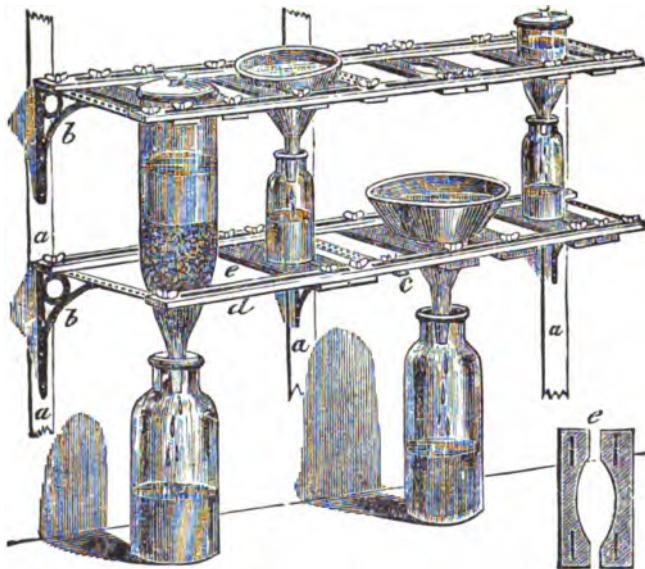


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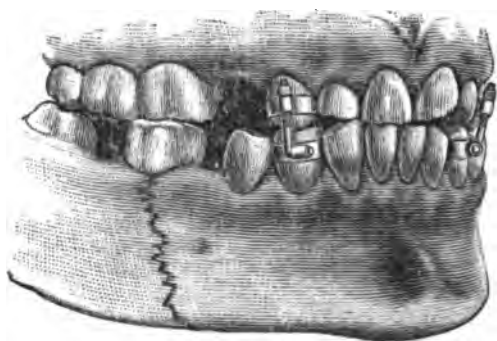
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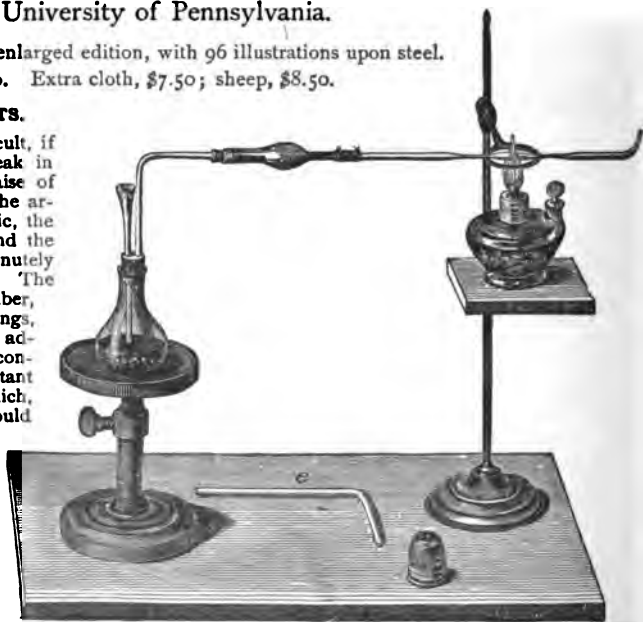
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